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THE ANATOMY OF THE CEREBELLUM.

By A. A. ABBIE,

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THE cerebellum must be regarded primarily as an organ whose fundamental concern is correlation of movement with proprioceptive impulse in the interests of bodily stability.

Proprioceptive impulses may be of two kinds: general from muscles, tendons and joints, and special from highly organized receptors. Special proprioceptors appear first in Ichthyopsida (fishes and amphibians) as a continuous line of organs extending from snout to tail along each side of the body. The system comprises the anterior lateral line system supplied by the seventh nerve, the vestibular apparatus supplied by the eighth nerve, and the posterior lateral line system supplied by the tenth nerve. In the terrestrial phase of amphibian existence, and in all higher vertebrates, the lateral line system disappears, only the vestibular apparatus being left; nor when higher animals (for example, reptiles and mammals) revert to an aquatic existence does the system recur. It is said (Kappers, Huber and Crosby[1]) that the cerebellum develops in relation to the central connexions of the lateral line system; when this disappears the cerebellum becomes devoted to the vestibular mechanism. Fixation of the cerebellum just behind the mid-brain is said to depend upon the attraction of higher, chiefly mesencephalic, coordinating centres.

The evolutionary history of the cerebellum has recently been reviewed by Herrick,[2] Kappers,[3] Kappers, Huber and Crosby[1] and Larsell.[4] The details are of no immediate concern here; it suffices to note that the cerebellum is represented almost throughout the vertebrate series. In *Amphioxus* and in the myxinoid cyclostomes there is no trace; in petromyzont cyclostomes J. B. Johnston[5] has recognized the beginnings of a cerebellum, and in all higher animals it is readily determined. In submammalia the organ appears to comprise a median, larger portion, with a smaller auricular lobe on each side; it is generally believed that in mammals the median part becomes the main cerebellar mass, while the auricles divide to form the flocculi and paraflocculi. Larsell,[4] however, believes that the auricles form only the flocculi, and that the paraflocculi are later developments. In an ascending phylogenetic scale the cerebellum increases in size and complexity while maintaining a uniformity of gross arrangement and minute structure. In mammals, with the sudden assumption of forebrain dominance, there is considerable expansion to the really massive structure familiar from the study of man.

In human embryology the cerebellum is forecast in the eighth week in the two lateral swellings which appear in the anterior margin of the rhombic lip. The swellings fuse in a dumb-bell-like structure which spreads transversely in front of the fourth ventricle. The mass becomes divided transversely by numerous fissures and sulci into as many lobules and folia. The cerebellum develops from the alar lamina of the neural tube, and its connexions remain predominantly of the somatic type. As is the tendency in suprasegmental structures (superior *corpora quadrigemina*, cerebral hemispheres), the majority of the neuroblasts migrate to the surface to form a cortex; some, however, remain behind as the various cerebellar nuclei.

External Appearance and Subdivisions.

The human cerebellum comprises two large rounded lateral parts, usually called hemispheres, connected by a median constriction generally known as the vermis. Projecting laterally, below the main mass on each side, are two lobulated processes—a larger flocculus and a smaller paraflocculus (Figures I, II and III). The larger trans-

verse fissures in the main mass are used to subdivide the cerebellum into a number of named units.

Sagittal section through the median constriction reveals the lobules so defined as: lingula, central lobule, culmen, declive, tuber, pyramid, uvula and nodule (Figure IV). The most important fissures employed are the uvulo-nodular (between uvula and nodule), primary (between culmen and declive), secondary (between pyramid and uvula) and prepyramidal (between tuber and pyramid). Both phylogenetically and ontogenetically the fissures appear in the order named; only the primary fissure completely subdivides the main cerebellar mass from side to side, while the uvulo-nodular fissure continues between the flocculus and parafloccculus on each side as the postero-lateral fissure.

Subdivision of the cerebellum into convenient units of morphological or functional importance has produced a number of diverse schemes (Kuithan,[8] Stroud,[7][8] Bradley,[3] Elliot Smith,[10][11][12] Bolk,[12] Edinger,[14] Ingvar,[15] Riley,[16][17] Abbie,[18] Larsell[4] and others). In the first place simple inspection reveals that the time-honoured separation into vermis and hemispheres cannot be sustained: the so-called superior vermis merges imperceptibly into the hemispheres (Figure I), while the inferior vermis so-called, while apparently discrete (Figure II), is equally inseparable on close examination. The subdivision serves no purpose save convenience, and when the terms vermis and hemisphere are employed here it is understood that convenience is the sole consideration.

Edinger proposed a distinction of palæo-cerebellum (vermis, flocculus and parafloccculus) and neo-cerebellum (the remainder). His views have been endorsed by Kappers,[9] Kappers, Huber and Crosby[1] and others, and have received a wide measure of support as a working hypothesis. However, collapse of the vermis as an entity reduces the force of Edinger's views considerably; moreover, Brouwer[19] and Biemond[20] have shown that the "vermis" is as much the recipient of the highest as of the lowest impulses, and so have further reduced any claim it may have to antiquity.

Elliot Smith[10][11][12] was the first to offer any reasonable morphological subdivision. Using the primary and secondary fissures, he distinguished three lobes—anterior, middle and posterior. Later, Bolk[12] recognized only anterior and posterior lobes separated by the primary fissure. Ingvar[15] modified Elliot Smith's conception by using the præpyramidal fissure to separate the middle and posterior lobes, thus shifting the pyramid from the posterior to the middle lobe. Riley[16][17] reverted to the bilobular conception of Bolk, and Abbie[18] has recently urged the merits of this simpler classification. Abbie's views were accepted by Elliot Smith, and subdivision into two lobes only, separated by the primary fissure, is now adopted as standard in English anatomical textbooks.

It was formerly the custom to subdivide the cerebellar hemispheres into a large number of lobes and lobules, but no useful purpose was served, and today the tendency is to restrict subdivision. The lateral extensions of the central lobule are called alæ, those of the culmen simply the lateral parts of the anterior lobe. The declive and tuber ("declive-tuber complex" of Abbie) extend laterally in the ansiform lobe which occupies most of the postero-superior and inferior aspects of each hemisphere; near the mid-line a prominent rounded part of the ansiform lobe stands out as the corresponding tonsil (Figure II). The pyramid, and possibly the uvula also, are said to be continuous on each side with the parafloccculus through a tenuous band of nervous tissue across the inferior medullary velum; the nodule is similarly continuous with the flocculi (Figure II). The reality of the nodulo-floccular connexion, at least, has recently been demonstrated for man by T. B. Johnston.[21] These slight bands are all that remain in man to mark the very stout connexions found in more primitive mammals.

Quite recently Larsell[4] and Larsell and Dow[22] have introduced a new conception in cerebellar subdivision. They state that the cerebellum is built around two commissures—vestibular and trigeminal. Around the vestibular commissure arose the flocculo-nodular lobe (nodule *plus* both flocculi), and around the trigeminal commissure arose the *corpus cerebelli* (remainder of the cerebellum). The separation is indicated by the uvulo-nodular and postero-lateral fissures; the *corpus cerebelli* is then subdivided according to the plan of Bolk, Riley and Abbie. In favour of this scheme are the following considerations: (*a*) the uvulo-nodular fissure is the first to appear in the cerebellar primordium (Elliot Smith); (*b*) the trigeminus system certainly makes great contributions to the cerebellum (Dart,[23] Abbie[24] and others); and (*c*) the nodule and flocculi certainly receive primary vestibular fibres (Ingvar[15]) On the other hand, Ingvar has shown that primary vestibular fibres also pass to the uvula and lingula, both of which are included in the *corpus cerebelli* by Larsell and Dow; until the discrepancy is disposed of the validity of this attractive scheme must remain in doubt.

Afferent Cerebellar Tracts.

Brief consideration of the kind of information which the cerebellum requires, provides a clue to the kind of connexions it must effect. Special proprioceptive impulses enter through the vestibular nerve. According to Ingvar direct vestibular fibres pass to the lingula in front, the uvula and nodule behind, and the flocculi on each side (the "basal ring" of Ingvar); in addition Dow[25] states that some pass to the fastigial nuclei. Indirect fibres from the vestibular nuclei pass to the same regions.

General proprioceptive impulses from muscles, tendons and joints enter the spinal cord through the posterior roots, and obviously every spinal nerve must contribute. In the cord the fibres follow two pathways—spino-cerebellar tracts and posterior columns. The fibres in the spino-cerebellar tracts are secondary from the posterior horns; the tracts are very ancient and originally formed a single bundle. In mammals the posterior tract is mainly direct, the anterior mainly crossed. The posterior column fibres are a comparatively recent addition, and should be considered of a higher grade. They ascend on the same side without interruption as far as the *medulla oblongata*. Here the posterior spino-cerebellar fibres, together with fibres relayed from the opposite posterior column through the gracile and cuneate nuclei, enter the cerebellum through the inferior cerebellar peduncle (*corpus restiforme*); they pass mainly to the pyramid and parafloccculi, but also to the uvula, culmen and declive (MacNalty and Horsley[26]). The anterior spino-cerebellar tract is also joined by secondary posterior column fibres (Mussen[27]) and it passes through the brain-stem to emerge on the surface of the isthmus immediately above the pons; here the tract turns dorsally, and back along the superior cerebellar peduncle (*brachium conjunctivum*) to enter the cerebellum (Figures V, VI and VII), where it is distributed chiefly to the central lobule, but also to the culmen and declive (MacNalty and Horsley). Within the spinal cord, and in the cerebellum, the spino-cerebellar tracts lie close together (Figures V and VI); their separation in the brain-stem of mammals results from interposition of the middle cerebellar peduncle (*brachium pontis*). Possibly this separation is more apparent than real, since Larsall[28] has described intermediate spino-cerebellar tracts.

It is known that large numbers of trigeminal fibres—both direct and indirect—enter the cerebellum (Dart,[23] Abbie,[24] Larsell[29] and others); indeed, both Dart and Larsell insist that the trigeminus provides one of the major primary components of the cerebellum. The destination of the fibres is uncertain; Dart, Abbie and Larsell have all observed the association of a large trigeminus with a large parafloccculus; Dart and Abbie correlate the two; Larsell, however, believes that the fibres go mainly to the anterior lobe. As opposed to Larsell's view may be adduced the fact that, although the trigeminus tends to be large in primitive mammals, in all except the Monotremata (Abbie[18]) the anterior lobe of the cerebellum tends to be small.

Large fibre tracts pass to the cerebellum from the inferior olives. The evolution of these organs has been traced by many workers, notably Kooy.[30][31] In man, each comprises a principal, purse-shaped mass and two associated nuclei—the medial and dorsal accessory olives (Figure VII). It is believed that the lips of the purse-

shaped portion, *plus* the accessory olives, constitute an older formation—palæo-olive—while the main mass of the principal olive represents a newer formation—neo-olive. The olivo-cerebellar fibres pass via the opposite inferior cerebellar peduncle (Figure VII); their destination remains uncertain, but Kappers[2] and Kappers, Huber and Crosby[1] state that the palæo-olive projects to the vermis, the neo-olive to the hemisphere. Certainly, the work of Holmes and Stewart,[31] Brouwer and Coenen[32] and Brouwer[33] supports the view that the greater part of the principal olive projects to the opposite cerebellar hemisphere; moreover, these authors find a very accurate point-to-point representation, both from side to side and from before back.

Other afferent fibres come from the reticular nuclei of the brain-stem and from the mesencephalic nucleus of the fifth nerve; but of these there is little accurate information.

So far only impulses from the medullary and spinal centres—mainly proprioceptive in nature—have been considered, but it is clear that whatever higher coordinating centre exists for muscular control must also send information to the cerebellum. In submammalia the forebrain is concerned chiefly, if not exclusively, with olfactory impulses, and the highest coordinating centre resides in the mid-brain—in the greatly hypertrophied "optic lobes" which represent the superior *corpora quadrigemina* of mammals. Thus the main descending tract is the tecto-cerebellar, which traverses the superior medullary velum. In mammals, transfer of sensory impulses other than olfactory to the cerebral hemispheres is accompanied by a progressive transfer of muscular control; thus cortico-cerebellar tracts arise. Primitive mammals reveal well developed tecto-cerebellar tracts (Hines,[24] Abbie,[24] Larsell[4] and others), but they dwindle progressively with increase in the cortico-cerebellar connexions; even in man, however, it is probable that some persist (Figure V). The advent of cortico-cerebellar tracts posed a serious problem: they descend with the pyramidal tracts on the ventral aspect of the brain-stem, the cerebellum lies on the dorsal aspect.

To achieve an association between cerebellum and the descending cortical fibres, cells from the embryonic rhombic lip migrate ventrally around the brain-stem. Each migrating cell plays out a fibre along its track, marking the course of its wandering and preserving communication with the cerebellum. Ultimately each cell comes to rest on the opposite side of the ventral mid-line in relation to a cortico-cerebellar fibre; thus each cerebral hemisphere is connected with the opposite cerebellar hemisphere. Naturally, the vast majority of the wandering cells collect immediately opposite the cerebellum as the large pontine nucleus, and their fibres are gathered in the mass of the middle cerebellar peduncle (Figure VIII).

While this is the fate of the majority of rhombic lip cells, others migrate elsewhere, and they produce the familiar external arcuate fibres which are often so abundant on the ventral and lateral aspects of the brain-stem (Figure VIII). The migrations of these cells have been followed by Elliot Smith[35][36][37] in particular. Most of the aberrant cells pass along the inferior cerebellar peduncle and come to rest in small masses called arcuate nuclei in the opposite pyramidal tract (Figures VII and VIII); thus their cerebellar connexions appear to join the inferior cerebellar peduncle. Some of the cells which entered the *nucleus pontis* pass dorsally and caudally beyond this, their fibres forming the *fasciculus obliquus pontis* (Figure VIII), and collect on the dorsal aspect of the inferior peduncle as the nucleus of the *fasciculus circumolivaris* (*corpus pontobulbare*). Yet other cells pass up to the mid-brain, or down to the spinal cord; even the so-called *striæ acusticæ medullares* are no more than aberrant external arcuate fibres. The wanderings are many and diverse, but the reason is uncertain; there may be some underlying neurobiotactic principle, although the inconstancy of pattern from brain to brain renders this unlikely.

Wherever the rhombic lip cells migrate, there the cortical fibres follow. To reach the arcuate nuclei cortical fibres pass with the pyramidal tracts beyond the pons until they encounter the cells to which they appertain (Figure VIII); and in the case of the nucleus of the *fasciculus circumolivaris*, the fasciculus itself is composed of cortical fibres which leave the pyramidal tract and pass dorsally behind the inferior olive to gain the nucleus (Figure VIII). As far as can be ascertained, every migrated rhombic lip cell becomes related to a cortical fibre, and apparently the connexion is always crossed.

As is well known, cortico-cerebellar fibres form two tracts. The fronto-pontine arises in the premotor cortex and descends in the medial third of the *basis pedunculi*, the temporo-occipito-parieto-pontine (temporo-pontine) arises from the parietal cortex between the sensory receptor areas and descends in the lateral third of the basis (Figure IX).

There seems to be little doubt that within the cerebellum the cerebral impulses are relayed mainly to the declive and tuber, and to the ansiform lobe (middle lobe of Ingvar, declive-tuber complex of Abbie). On morphological grounds Abbie[23] considered that the fronto-pontine fibres end mainly in the front of the pons and relay to the declive-tuber complex, while the temporo-pontine fibres end mainly in the back of the pons and relay to the culmen and related parts of the anterior lobe (Figure IX). Sunderland's experimental investigation[38] has largely confirmed this; but Sunderland was unable to trace any connexion between culmen and pons. Against his single negative finding, however, must be set the whole of the morphological evidence, and the clinical observations of Brouwer[19] and Biemond.[20] These authors describe a case of localized infarction at the caudal border of the pons associated with degeneration in the anterior lobe of the cerebellum, particularly the culmen. It may be assumed that Abbie's views offer a reasonably close approximation to the truth.

Thus the cerebellum may be regarded as a complex of successive neural levels: firstly, vestibular and, possibly, trigeminal; secondly, spinal; and thirdly, cortical. It is clear that there is considerable overlap between these levels (Figures X and XI), and it would appear that the cortico-cerebellar system has been imposed upon a more primitive vestibulo-spinal.

Minute Structure of the Cerebellum.

As mentioned previously, except for those which remain behind as the cerebellar nuclei, the cells of the cerebellum collect at the surface as a cortex, and this cortex is vastly expanded by multiplication of the folia. Many workers have contributed to our knowledge of cerebellar histology, but none more than Cajal,[39] and his account is followed here.

The cortex presents an inner granular layer and an outer molecular layer. The granular layer contains multitudes of "granule" cells whose axones pass vertically into the molecular layer, there to dichotomize—the branches passing in opposite directions along the axis of the folium (Figure XII). The term molecular layer comes from the appearance of these branches cut across. The molecular layer contains many cells, the most outstanding being the large Purkinje cells which form the deepest stratum. The Purkinje axones pass down into the white matter, while the dendrites are arranged in the familiar ivy-on-the-wall manner transversely to the longitudinal axis of the folium, disposed as a series of sieves in respect to the granular axones (Figure XII). Of the other cells in the molecular layer the most important are the "basket cells" whose processes run transversely across the folium linking the Purkinje cell bodies by basket-like investments (Figure XII).

Of the many kinds of fibres ascending to the cerebellar cortex, most striking are the "mossy" and "climbing" fibres. The mossy fibres divide to end in relation to several granule cell dendrites; thus their impulses are widely diffused through the folium by a number of dichotomized granular axones (Figure XII), in relation to a number of Purkinje cells. The mossy fibres wind around the axones and dendrites of the Purkinje cells and so receive individual representation. Accordingly, it is believed that mossy fibres carry spinal impulses, climbing fibres cerebral impulses. The mode of termination of other ascending fibres is uncertain, but it is probable that

the vestibular fibres, at least, end in direct relation to the Purkinje dendrites.

Efferent impulses from the cerebellar cortex are carried by Purkinje axones. It is possible that some go directly to the vestibular nuclei (Clark and Horsley[40]); Dow[23] believes that the nodule and flocculi, and possibly the uvula also, send fibres to the vestibular nuclei. Apart from this most of the Purkinje axones appear to impinge upon the cerebellar nuclei. Generally, four nuclei are described on each side; from medial to lateral they are: fastigial (tectal), globose, emboliform (interpositus) and dentate (Figure XIII). The fastigial and globose constitute an older pair whose impulses probably come from the uvula, nodule and pyramid (Clark and Horsley, Dow). The emboliform and dentate nuclei are newer formations; the emboliform appears to receive fibres from the culmen, uvula and paraflocculus (Dow), the dentate from most of the remainder of the corresponding cerebellar hemisphere.

Efferent Cerebellar Tracts.

Apart from the direct efferent fibres to the vestibular nuclei already mentioned, cerebellar efferents come principally from the cerebellar nuclei, and they fall into two groups. Some fibres from the fastigial and globose nuclei cross at once in the cerebellum and pass around the fourth ventricle and under the floor to join opposite the medial longitudinal fasciculus; other fibres pass around the same side of the ventricle and end in the homolateral vestibular nucleus (of Deiters). Thus the vestibular part of the cerebellum relays partly to brain-stem nuclei along the medial longitudinal fasciculus, and partly to spinal centres through the vestibulo-spinal tract which arises from the lateral vestibular nucleus (Figure XIII). As the fibres from the medial pair of nuclei pass around the fourth ventricle they form a hook-like bundle known as the *fasciculus uncinatus* of Risien Russell.

The efferent fibres from the emboliform and dentate nuclei collectively give rise to the superior cerebellar peduncle which plunges downwards and forwards into the mid-brain (Figures IX and XIII). Here—under the inferior *corpora quadrigemina*—the peduncles decussate, and under the superior *corpora quadrigemina* some three-quarters of the fibres end in the opposite red nucleus.

The red nucleus is a greatly hypertrophied reticular nucleus (Kappers, Huber and Crosby), which in man and many of the higher animals comprises two portions: a posterior large-celled and an anterior small-celled. The older large-celled part (palæo-rubrum) gives rise to the corresponding rubro-spinal tract; the tracts of the two sides decussate in the mid-brain (ventral tegmental decussation, decussation of Forel) and pass back along the brain-stem to the spinal cord (Figures IX and XIII). The small-celled part is much newer (neo-rubrum) and attains its greatest size in man. It gives rise to the rubro-cervico-spinal tract which passes to the upper part of the spinal cord.

The one-quarter of the superior cerebellar peduncle which does not end in the red nucleus passes on to the diencephalon (Figure XIII), there to end in one of the thalamic nuclei; but which one, remains in some doubt. Le Gros Clark[41][42] believes it to be the arcuate nucleus which projects to the foot of the precentral gyrus and the cortex immediately in front. Earl Walker,[42][44] on the other hand, places the termination of the peduncle in the nucleus *ventralis lateralis*—the anterior parts of the ventral and lateral nuclei—whence the projection is to the precentral gyrus.[1] At all events, it is clear that cerebellar impulses do relay through a thalamic nucleus to the precentral gyrus.

Bodily Representation on the Cerebellum.

The problem of representation of parts of the body on the cerebellar cortex possesses great potential clinical importance. A priori, the uniformity of cerebellar structure throughout might be urged against such a conception, but it cannot be adduced as positive evidence. Bolk[13] studied the question by noting localized cerebellar developments in animals with specialized motor function. The method is quite valid, but unfortunately most of Bolk's conclusions have failed to withstand critical analysis. He made the interesting observation that the paraflocculi are greatly enlarged in cetaceans (for example, whales), and Dart[28] noticed the same in the extinct Zeuglodontidæ (which were probably cetaceans). Bolk endowed the paraflocculi with a special balancing function, but they are equally large in such a non-aquatic animal as the echidna (Abbie[26]). Larsell[4] believes that the paraflocculi have a special proprioceptive function, but there is little positive evidence at present.

There is a general belief that the anterior part of the body is represented in the front of the cerebellum, the posterior part behind, and the extremities in the hemispheres. Bound up with this is the view that olivary enlargement reflects increasing control over the musculature of the extremities. Larsell[4] believes that the anterior lobe of the cerebellum is concerned with the musculature of the head and snout, and that the trigeminous system is involved there also. It has already been shown, however, that the anterior lobe is generally small in primitive mammals, despite a large trigeminus. Only in higher mammals, and particularly primates (Abbie[13]), is there any notable enlargement; and this enlargment cannot logically be dissociated from the concomitant increase in cerebral connexions. The problem of bodily representation on the cerebellum remains obscure.

Cerebro-Cerebellar Relationship.

It is clear that in mammals cerebellar expansion has proceeded more or less *pari passu* with cerebral expansion, and the cerebro-cerebellar connexions have increased in accordance. The relationship is not solely of morphological interest; its importance has been shown by experimental (Weed,[46] Warner and Olmsted,[47] Bernis and Spiegel,[48] Fulton,[49] Earl Walker[50] and others) and clinical observation. From the evolutionary point of view the importance of this growing relationship in man's acquisition of muscular skill has been emphasized by Elliot Smith[51] and Abbie.[13] The relationship, however, does not appear to be absolute. Putnam[52] has shown that while in general cerebellar increase runs roughly parallel with the evolutionary scale in mammals, those which display special motile capacity (for example, the elephant, sea-lion, cat, bear, anthropoids) betray a cerebellar proportion above the average. Thus the cerebellum in the great apes is relatively larger than in man. But, no doubt, this apparent falling off in an animal with such dexterity as man must be ascribed largely, if not entirely, to the enormous expansion of the human cerebral hemispheres.

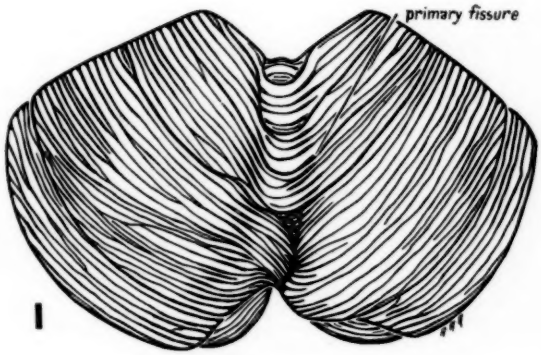
Conclusion.

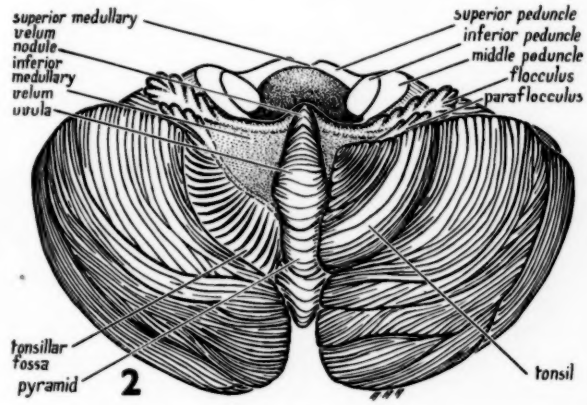
Throughout the vertebrate series persistence of the most primitive cerebellar connexions indicates a persistence of the original equilibratory function. The vestibular mechanism of the internal ear reveals a fairly progressive improvement in submammalia, but little in mammals. In the submammalia the higher, tecto-cerebellar, connexions do not appear to increase notably, so that submammalian cerebellar expansion may be attributed largely to increase of vestibular connexions. In mammals, however, vestibular increase, if it occurs, is relatively slight—certainly insufficient for the sudden and progressive cerebellar expansion which is so striking. The only new factor concerned is the appearance and expansion of cerebral connexions, together with a few associated mechanisms such as the inferior olives.

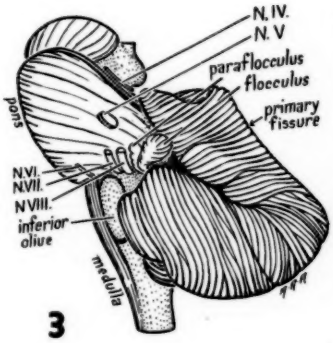
There can be little doubt that the cortical connexions are associated with cortical control of muscular movement; origin of the cortico-cerebellar fibres from the premotor cortex, and from the cortex between all the sensory receptor areas, is a clear reflection of this fact. The progressive increase in cortico-cerebellar connexions argues an equally progressive increase in cortical control of movement; this is supported by experimental and clinical observation, and the increase is greatest in man.

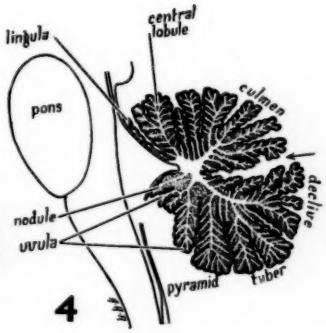
[1] In a previous communication (Abbie[45]) Earl Walker was incorrectly credited with belief in the arcuate nucleus as the thalamic intermediary: Earl Walker's views are as stated here, and I must apologize for the error.

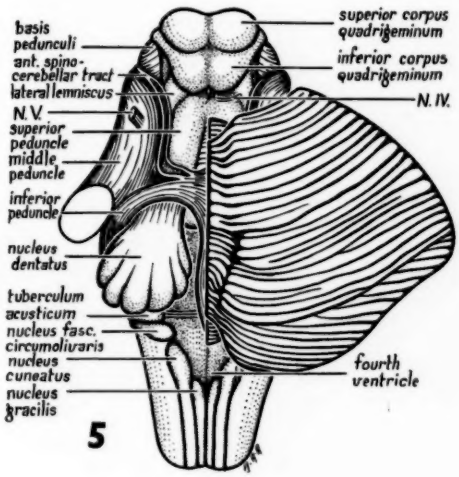
ILLUSTRATIONS TO THE ARTICLE BY DR. A. A. ABBIE.

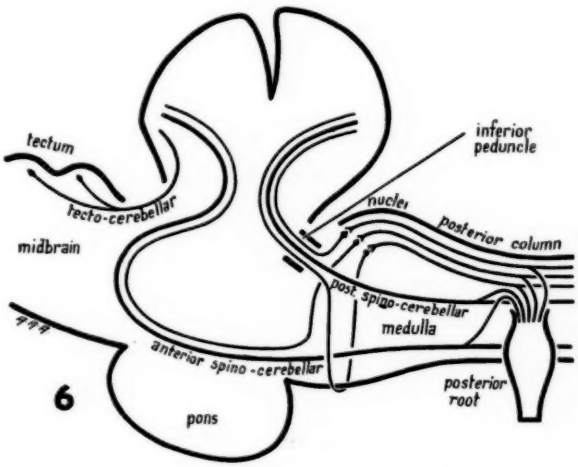


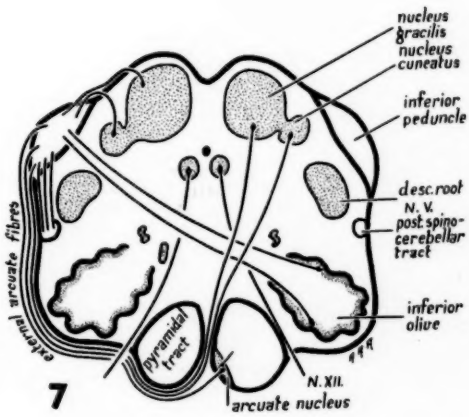




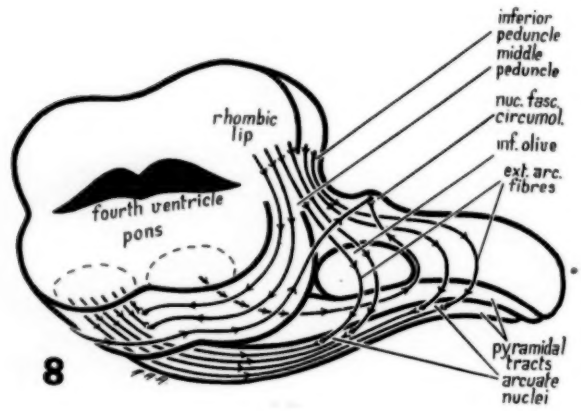








ILLUSTRATIONS TO THE ARTICLE BY DR. A. A. ABBIE.



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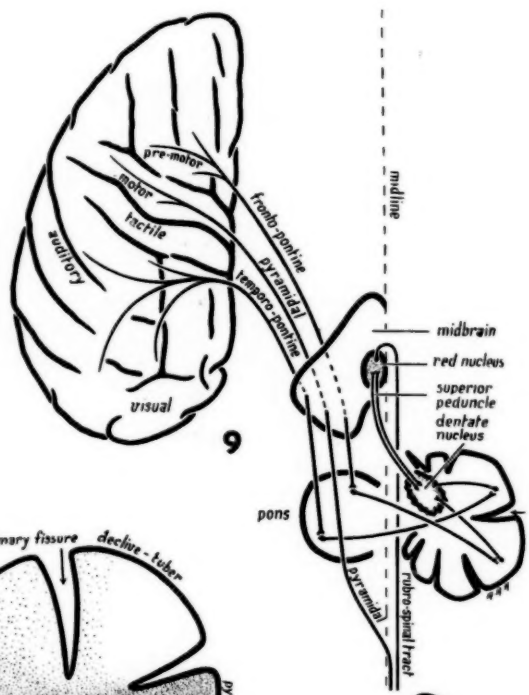

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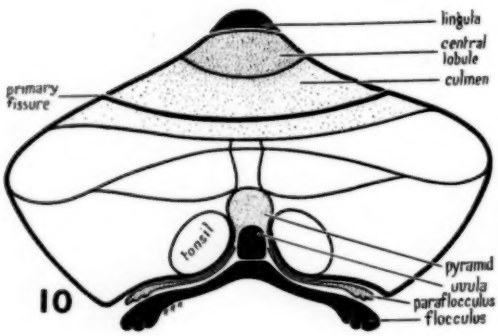

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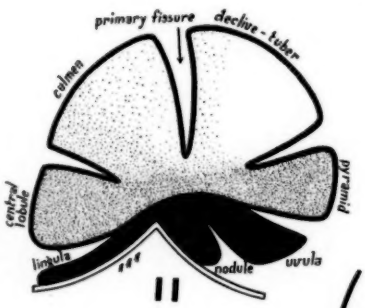

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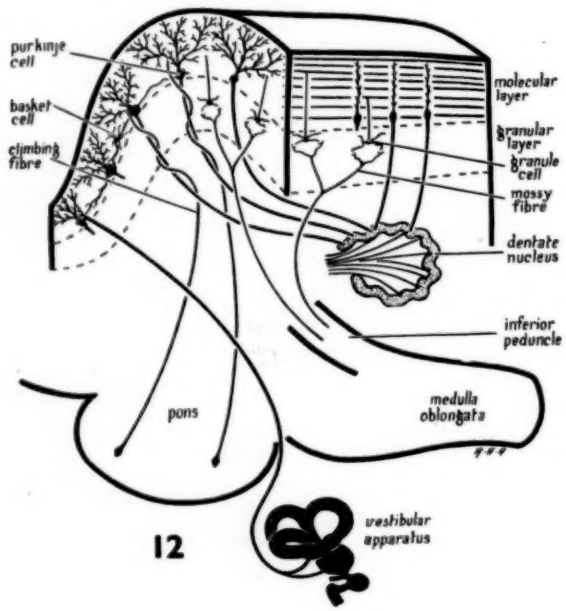

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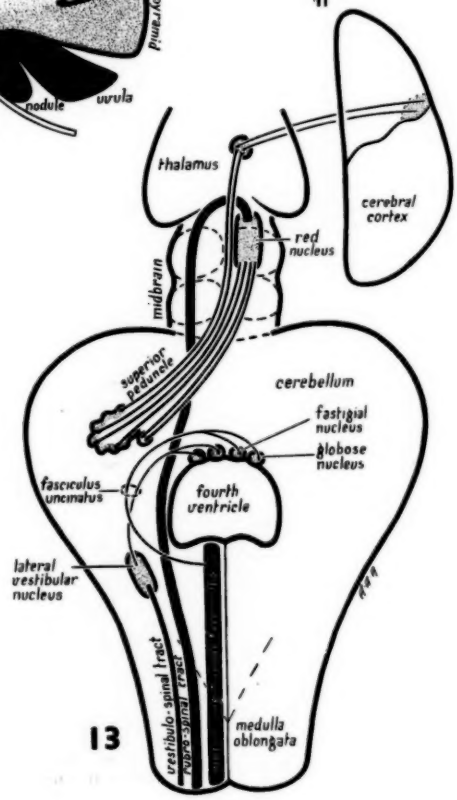

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The story of the cerebellum bespeaks two distinct phases in evolution. In the earlier there is emphasis on bodily stability as determined by correlation of movement with proprioceptive information; there is probably some muscular coordination for movements mediated through the mid-brain also. In mammals the equilibratory gain is consolidated; then, transfer of muscular control to the cerebral hemispheres releases the motor mechanism from the rigid dominance of the mid-brain: a more flexible and adaptable system is substituted and the potential range of muscular movement widens vastly. The increase in muscular skill and the increase in cerebro-cerebellar interplay proceed progressively in the ascending mammalian scale and attain a climax in man; to them man owes his exquisite preeminence in work, art and sport.

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Legends to Illustrations.

(See special plate.)

FIGURE I. The cerebellum from the dorsal aspect; note that the "superior vermis" cannot be distinguished from the "hemispheres".

FIGURE II. The cerebellum from the ventral aspect; the right tonsil (on the left side of figure) has been removed to expose the inferior medullary velum and the connexions of the flocculus and paraflocculus with the nodule and pyramid respectively.

FIGURE III. Left lateral aspect of the cerebellum; note the flocculus and paraflocculus.

FIGURE IV. Median sagittal section through the cerebellum to show the major subdivisions.

FIGURE V. Exposure of the peduncles of the left cerebellar hemisphere; partly schematic—based on two dissections.

FIGURE VI. Schema to illustrate the course of the spino-cerebellar and posterior column fibres to the cerebellum.

FIGURE VII. Schema to illustrate the components of the inferior cerebellar peduncle (*corpus restiforme*).

FIGURE VIII. Schema to illustrate the migrations of rhombic lip cells to meet cortico-cerebellar fibres. (Modified from a figure by Elliot Smith.)

FIGURE IX. Diagram of cortico-cerebellar connexions. (After Abbie.[18])

FIGURE X. Diagram (modified after Ingvar) to illustrate the main fibre projections in the cerebellar cortex. Black = vestibular, stippled = spinal, plain white = cerebral.

FIGURE XI. Schematic sagittal section to show the cerebellar levels. Black = vestibular, stippled = spinal, plain white = cerebral. (After MacNalty and Horsley, Ingvar, and others.)

FIGURE XII. Schema of a cerebellar folium to show its main features. (Mostly after Cajal.)

FIGURE XIII. Schema of the main efferent tracts from the cerebellum.

SOME NOTES ON THE PATHOLOGY OF THE CEREBELLAR SYSTEM.[1]

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THE Section of Neurology, Psychiatry and Neuro-Surgery having chosen the cerebellum as a subject for discussion, has also arranged four aspects of approach: the anatomical, the physiological, the pathological and the clinical. This arrangement relieves the pathologist of many paragraphs.

The word "ataxia" has for too long dominated all ideas associated with the cerebellum, including its pathology; indeed, many older text-books on neurology dismissed this aspect with a few lines. Symptoms of ataxia, hypotonia and so on at once called up the cerebellum, in spite of the well-known pathology of Friedreich's ataxia, which in its simplest form need involve only certain tracts of the spinal cord—the spino-cerebellar tracts, the posterior column and sometimes much later the cortico-spinal tracts. Of course, it might be assumed the various cerebellar tracts in their degenerated state would ultimately form a pathological tract in the inferior or superior peduncle; but even this might not hold. Degenerated myelin has a habit of stopping at the top of the cord, as in the myelitides, and cases in which widespread degeneration of olive cells and pontine neurones obtained have been associated with a relatively normal restiform body and middle peduncle. For this reason the late A. W. Campbell felt constrained to invoke vascular failure for part of the pathology of Friedreich's ataxia. In any case, but little abnormality would be found in the cerebellum. Of course there are forms of this disease with much wider involvements including the cerebellum itself, and in siblings *formes frustes* occur with other stigmata of degeneration, such as mental deficiency and absence of some organ or function. Clinicians, having had the luck to strike a series of these rare cerebellar symptom complexes, proceeded to tabulate them with the poorest accompanying pathological investigations, often without any. Kinnier Wilson reminds us that "ataxic" symptoms have *post mortem* been found to be unaccompanied by cerebellar abnormality, and conversely, an undersized and even grossly diseased cerebellum has caused no corresponding symptom complex during life. Finally, a study of the connexions of the cerebellum will show that lesions anywhere in that organ may produce "cerebellar" symptoms.

The Pathological Problem.

The complete examination of any brain submitted to this laboratory has always included the cerebellum, the top of the spinal cord, the medulla with olive, the pons and the peduncles of the cerebrum. It is pleasing to note that the Werthams (from whose manual I shall borrow freely) recommend this, because, since the anatomical histology of the cerebellum is so simple, pathological phenomena in that area are more easily interpreted than in the cerebrum, and they are alike in their reaction to disease.

The reward of workers in this laboratory has been to note that the cerebellar pia is often better preserved than the cerebral pia, and this has revealed signs of hæmorrhagic, leucocytic, or lymphocytic invasion, as well as pial hypertrophy, œdema, specific infections like tuberculosis and even extensions of tumour invasion from elsewhere. More rarely we have noted intracerebellar hæmorrhages and softenings seen with the naked eye or microscopically. Once a tuberculoma had been shelled out like a bean (giant celled systems and bacilli were revealed) by a surgeon who refused our diagnosis, desiring that of a glioma.

In spite of modern observations on Purkinje cell changes, I must confess with Mott that I have generally found these cells well preserved. The cerebellum has often been found of quite decent proportions and healthiness when the rest of the brain has shown grievous disproportion and damage. However, I cannot say this of the dentate nuclei, because the neurones there are more vulnerable than even those of the olives and the sommer sector of the hippocampus. Extreme degrees of axonal degeneration (clinically this is not often important), great chromatic variations and even outfalls, are often evident, while in the dentate capillaries both incrustations and arteriosclerosis are often observed. The white substance, too, is an excellent place to look for hypertrophied glia cells, perivascular gliosis and argentophile plaques; once a profound gliosis in that area clinched the diagnosis of encephalitis in the case of a workman who had fallen in a fit, when the cerebral pathology was none too clear. Productive arteritis can be well studied, too, in the cerebellar white matter, and of course a Purkinje cell may show altered shape and staining qualities, atrophy or even dropping out. To prove the last phenomenon since these cells are not regularly and evenly spaced out, one uses Cajal's silver stain, by which the hardier basket cells and processes very often surviving may reveal baskets empty of Purkinje cells. Many cerebellar cysts have come to us; but their imperfectly fixed, distorted and squashed condition has often precluded differentiation between some developmental state and some liquefying astrocytoma or other glial hypertrophy—a state of affairs common in the cerebellum and in the young.

The Cerebellar System.

Apart from some general diseased states just enumerated, certain more or less rare syndromes have intrigued clinicians; these are mostly atrophic phenomena, sometimes pure, sometimes grafted on an agenesis, and sometimes (as hinted by Parker and Kernohan) the aftermath of encephalitis, as happens in "louping ill" of sheep. Classification has been attempted on the basis of the part or parts involved pathologically. These will now be set out in detail.

A start is made with the cerebellum, and this is examined as a whole for size, shape and development; its pia or the molecular layer is examined for contained superficial glia, Bergmann's fibroglia cells and the golgi Type II cells. Then the Purkinje neurones themselves are examined to determine whether these show a centripetal or a centrifugal type of degeneration. The centripetal type of degeneration, which tends to obtain in diffuse conditions like amaurotic family idiocy and illnesses generally, implies swelling of the antlers or plumes (leucocytes being found on them in the early stages) and axis cylinder enlargements (torpedoes), and is commonly accompanied by degeneration of the basket cell system. The centrifugal form of degeneration, which is more common in the heredo-cerebellar types, is that in which the processes of the Purkinje cells disappear, as does the rest of the neurone gradually, while on the other hand the basket-cell systems not only survive, but appear to hypertrophy or at least thicken. The Bergmann glia cells also hypertrophy (a sort of substitution hypertrophy); in extreme cases they may send out dense vertical processes, which may drag down the pia in sclerosis. The granular layer is examined for general or patchy thinning of neurones, their condition and the moss and climbing fibres therein being noted; the next step is to the molecular layer, as well as collaterals from the Purkinje cells. In intoxications like lead encephalopathy capillaries in this region show grave incrustations. The medulla or white substance is noted for its size; it may show gross atrophy or distortion, which may be due to agenesis, myelitis, sclerosis, macroscopic or microscopic hæmorrhages or softenings. It is at times swollen, as in encephalitis. The dentate nuclei (with which may be examined the other nuclei) often show degeneration or capillary incrustations; the amount is noted. The peduncles rarely contain some special lesion like a tumour, softening or other damage; more often examination merely reveals some tracts injured at some other source. A Weigert-Pal section of the mid-medulla reveals the state of the olive clls, gliosis in that region, infarcts, the nature and number or myelin fibres entering or leaving the hilum, or other aspects of the olives or other important medullary nuclei—the restiform bodies and vestibular apparatus. Also one notes the olivo-cerebellar fibres, the thalamo-olive fibres and the arcuate

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neuro-Surgery of the New South Wales Branch of the British Medical Association on July 22, 1940.

nuclei and fibres, the pons for its intrinsic nuclei and transverse fibres to the middle peduncle, and the brain stem for tracts coming from and making to the *brachium conjunctivum*. Finally, a myelin-stained section of the spinal cord reveals any tract degenerations there; it is said that in the Marie type the ventral tracts are more often affected than in Friedreich's ataxia. Further cerebellar connexions need not detain us. A lesion in one or more of these cerebellar connexions may cause ataxia; the cerebellum may be involved as well, or in one type it may be involved (ideally) alone. On these conceptions one may proceed to the classification.

The Cerebellar Classifications.

Jelliffe and White commence by discussing lesions in the various peduncles and then so-called lesions within the cerebellum, and classify them in the following way: (i) pure unilateral ageneses and atrophies, (ii) pure bilateral ageneses and atrophies, (iii) cerebellar atrophies associated with (*a*) disease of the cerebrum, (*b*) disease of the spinal cord. The first and second categories explain themselves; (iii)*a* is considered in detail, but not with any clarity. Many of the patients so affected are idiots. The cerebellar-cerebral types possibly include Gordon Holmes's "congenital smallness of the central nervous system with cerebellar symptoms". A type called "olivo-pontine-cerebellar atrophy", well described by Thomas, may be mentioned here. Of this Kinnier Wilson writes: "It shows a fairly definite syndrome." There is atrophy of the cerebellar cortex, of the bulbar olive and of the grey matter of the pons. Total degeneration of the middle peduncles of the cerebellum and partial degeneration of the inferior ones, and a relative integrity of the cerebellar nuclei are present; the lesion is usually in old age and progresses slowly. A fair example of the confusion engendered by the inclusion under the caption "Marie's hereditary cerebellar atrophy" of states with a wide pathology occurs here. This group is recommended for inclusion both here and in (iii)*b*. It is said to be allied to the olivo-pontine-cerebellar type. Kinnier Wilson talks of an "olivo-cerebellar" group; but a few examples only have been noted and even these he would subdivide. The areas chiefly involved are the olivary system and some part of the cerebellar cortex. Such are Holmes's cases of "familial degeneration of the cerebellum" in siblings with sexual under-development. But Holmes elsewhere notes that with extensive and long-standing degeneration of the Purkinje cells, the olive cells likewise degenerate—a sort of tertiary or retrograde degeneration—and this picture quite resembles the findings in a case of ours which I shall discuss later. Gross destruction of one cerebral lobe may be associated with a similar condition contralaterally.

Group (iii)*b* concerns involvement of the cerebellum and spinal cord; it is a motley group. Suppose we allow ourselves to group here both Marie's and Friedreich's types, then we shall have every gradation from the Friedreich type, with much cord and little cerebellar damage, and to Marie's, with more cerebellar and little cord change. Kinnier Wilson, discussing the latter type, calls it "spino-cerebellar atrophy", and states that it is characterized by "a general hypoplasia of the cerebellum in whole or in part", and then of the pons, medulla and cord. Cerebellar lesions would imply a small organ with involvement of all tissues and also dentate and olive neurones; add to this wasting of superior peduncles and transverse ponto-cerebellar fibres. Three sets of combinations of lesions are given for different families; many more are surely possible. Perhaps they stand midway between Friedreich's and the olivo-ponto-cerebellar types.

Primary Parenchymatous Degeneration.

Quite by itself is noted "primary parenchymatous degeneration" or chronic progressive degeneration of Purkinje cells. Holmes has described this condition and Kinnier Wilson includes it in a class under the heading "intracerebellar and cerebellofugal types". This is the "parenchymatous cortical cerebellar atrophy" of Parker and Kernohan. An excellent case is to hand from Akelaitis, of Rochester, New York, in which hereditary features and cerebral involvement were present. Our laboratory must be congratulated on having obtained a similar case (of a type of which less than 20 perhaps have been described to date) from the clinic of Dr. Kenneth Noad and Dr. Bruce Hall, together with an excellent family history and clinical details of many members. The main pathological process is a Purkinje cell atrophy and outfall of the cerebello-fugal type, with hypertrophy of the Burgmann fibroglia and remarkable outstanding prominence of the basket system and preservation of almost all the other neurones. However, here must be mentioned another pitfall—the question whether the distribution of the Purkinje atrophy is universal or apparently confined to, say, the deep recesses of the folia or even to the neocerebellum. But controversy exists on what constitutes the boundaries of the palæo-cerebellum and neocerebellum, and Brouwer, who once maintained this distribution for the olive and cerebellum, has since changed his ideas. In Parker's case the superior vermis and quadrilateral lobes were most affected; in Akelaitis's case the *lobulus centralis* was most affected, and the process grew progressively less backwards over the culmen and quadrilateral lobes; whilst in our case universal and equal atrophy and outfall were present. Again, in Parker's case the affected areas stopped abruptly and the cerebellum alone was affected. In Akelaitis's case cerebral cortical outfalls were present; the serial sections of the brain stem revealed only olive damage, which was worst in the medial portions of the dorsal layer of the inferior olives, also in the dorsal and medial accessory olives. Some loss of fibres obtained in the olivo-cerebellar tract and in the cerebellum, with cerebello-fugal Purkinje cell atrophy and loss. All the central nuclei were normal. Gliosis was pronounced. This observer also reports another case clinically and pathologically very similar. In both cases the spinal cords were normal. Thus cases in which in addition the medulla or olivary system is affected have been discussed, and also the question arises whether other systems like the cerebrum are involved as well. In our case the loss of Purkinje cells was both universal and almost entire. The outfall of olive cells was most extensive and evenly spread. Medullary fibres from the hilum *et cetera* and in the olivo-cerebellar tracts were grossly reduced, and careful examination above and below the medulla and the peduncles revealed no other lesions save the accompaniments of arteriosclerosis. Lastly, pure cerebello-fugal types implying lesions involving affections of tracts to the red nuclei are postulated, but await a better knowledge of these tracts. Other questions raised include: (i) To what extent do these atrophies resemble Pick's disease of the cerebrum? (ii) Do Purkinje cells tend to age early—a sort of Altzeimers presenile dementia of the cerebellum? In a case of Friedreich's ataxia described from this laboratory the main Weigert-Pal degenerations in the cord stopped at the medulla. A little paling was observed in the restiform body, but only Marchi preparations permitted the observation of isolated affected myelinated fibres, such as those in the mesial fillet, external arciform fibres, restiform body and post-central gyrus. The cerebellum was normal save for some neurones in the dentate nuclei, where some gliosis obtained. A sibling was also affected similarly, according to this communication.

Obvious Developmental Conditions.

The average weight of the cerebellum is 150 grammes; 10% is added if the specimen is fixed in 10% formalin solution. Considerable variations in weight are compatible with normality. In idiots the cerebellum may be absent, represented by a mere sheet of structure, and this state is compatible with long life. In a cyclopian pig I found the cerebellum represented by a small hump, as in the skate; transverse section revealed about five tiers of the three cerebellar layers and general hypoplasia. Two Newcastle idiots, with one cerebral hemisphere shrunken and an absence of pyramidal system there, had the cerebellar hemispheres on the contralateral side similarly reduced; while of two other idiots, one with both cerebral hemispheres and the other with one consisting merely of cysts, the former had quite a good cerebellum while the latter had the contralateral cerebellar hemisphere almost destroyed. In one of a family of four siblings referred to

me by Dr. Nowland, all showing cerebral sclerosis, probably 60% of the Purkinje cells were missing and the remainder swollen and pale. It has been suggested that in true congenital disorders the phylogenetically younger parts are the more involved. However, many deny any special significance in the distribution of the atrophy in acquired cerebellar atrophies. Cerebellar disturbances may be expected in cretinism, tuberose sclerosis, microgyria and hydrocephalus, in Tay-Sachs disease and the more adult type, Batten-Mayou's disease. The associated degeneration of the olives, the arcuate system and the pontine neurones may be due to their common origin from a group of budding neurones, developing from the lip of the fourth ventricle (the rhomboid fossa), thus constituting an abiotrophic involution. Commonly in these children fewer Purkinje cells obtain, and these may be more or less dislocated and the layers may run into one another. Poor development is associated with atrophy; indeed, the latter may be superimposed on the former, and *lues* may further complicate matters. Substitution gliosis is added, and rarely, the Golgi Type II neurones and the basket system may also be involved. A great degree of retrograde atrophy is nowadays recognized, and may gravely simulate an agenesia. Lastly, in fœtal states (to the ninth month) there obtains an outer granular layer in the molecular layer. This at times persists. Is there any need to attempt to classify these defections?

The Golgi cells are lipophylic, the Purkinje cells the reverse; so care must be taken with fat stains, and double nuclei are not necessarily "a find". It may be added that the dorso-median (microgyric) portion of the dentate nucleus is old, and its latero-ventral (macrogyric) part is new, and from these two divisions of any structures so divided definite fibres pass to palæo-structures and neo-structures elsewhere.

Vascular Phenomena.

Gross cerebellar hæmorrhage is uncommon, but areas up to a centimetre in diameter may be encountered in certain infections like typhus fever and influenzal states. Minute yellow brown softenings of individual folia may be seen in arteriosclerosis and states of hypertension corresponding to the relatively large areas in the cerebrum. R. M. Norman describes the results on the brain, peduncles and cerebellum of thrombosis of the superior longitudinal sinus. Certain parts of the cerebellum and not others were picked out for damage. For instance, examination of the superior vermis revealed almost complete loss of Purkinje cells, and those surviving showed atrophy on a cerebellopetal basis. However, the rest of the histo-pathological picture was on a cerebellofugal basis, to be discussed later. Tangential glia and fibrosis are not natural in the outer or molecular layer, but after certain vascular failures a mesoblastic sclerosis of this nature may be found there as in the cerebrum. The presence of many leucocytes on Purkinje plumes in the early stage of degeneration presages anoxæmia rather than infection. Other interesting phenomena include partial atrophy of the tongue in occlusion of the inferior cerebellar artery. Dr. Abbie kindly called my attention to an article by A. Biemond, picturing a lesion in the distal part of the pons, nearly mesial, apparently procuring a contralateral degeneration in the cerebellar cortex anteriorly and likewise near the mid-line. One should note that regions quite devoid of Purkinje cells are bordered by folia with partial denudation before healthy folia again obtain. Sometimes these partial degenerations are explained as retrograde. Mills mentions a case in which a thrombosis of the superior cerebellar artery was associated with a lesion extending to the dentate nucleus.

Infective States.

Many infections common to cord and brain likewise affect the cerebellum—tuberculosis, encephalitis (*encephalitis lethargica* and Medin's type and encephalomyelitis), *lues*, general paresis, chicken pox, typhus fever, malaria, whooping cough, hydatid disease, torula, rabies, the miliary abscesses of puerperal fever, and naturally infective states associated with the fourth ventricle, abscesses from middle ear disease, wounds and trauma, long antedated and metastatic from the cord. Allusion is made to louping ill in sheep and equine encephalomyelitis, as they resemble human states; moreover, rare cerebellar syndromes are made clearer by the study of similar states in animals, in which, too, may be found examples of disseminated sclerosis and Schilder's disease. Tuberculomata and gummata at times resemble tumours. The reactions of the various glial elements are as in the cerebrum; but mention should be made of the "shrubbery" formation in the molecular layer, an acute reaction; when it is microglial it commonly follows the plumes and eventually disintegrates, leaving nuclei behind. Marginal fibrous glia, of doubtful importance at times in the cerebrum, is always abnormal in the cerebellum and of positive import. To be carefully distinguished from infective consequences are those lesions which result from intoxications by alcohol, lead, phosphorus, mercury, snake and spider venom, veronal, phosgene, "Salvarsan", narcotics *et cetera*. Also distinguish the glia nodules, aftermath of minute hæmorrhages in blood diseases like pernicious anæmia, leuchæmia and scurvy. However, these can result from influenza and sepsis.

Tumours.

Tumours of the cerebellum may be vascular or meningeal (and possibly derived from these), psammomata, cholesteomata, endotheliomata, lipomata and myxomata. Then we have gliomata, ependymomata, medulloblastomata, ganglioneuromata and secondary metastases from elsewhere. Cerebello-pontine angle tumours, chiefly schwannomata from the eighth nerve, naturally affect the cerebellum from contiguity, as may also tumours in the occipital lobe and those associated with the fourth ventricle. Not all angle tumours are schwannomata, for I have met in that region glioblastomata and meningioblastomata. Once also a secondary deposit from the breast had raised a lump 1·0 centimetre high near the *striæ acusticæ* indenting the cerebellum. A tendency of cerebellar glial tissue to become liquid has been noted, when the walls may still be formed of glia and the tumour element confined to a nodule at one part; this last part must be removed to effect complete extirpation. Angioblastomata and medulloblastomata are more common in the cerebellum in youth and in the cerebrum in adults; the reverse holds for astrocytomata. Lindau's angioma in the cerebellum associated with retinal growths may derive from embryological vascular mistakes above the medulla.

Special Stains and Impregnations.

Paraffin sections from formalin tissues may be used with hæmatoxylin-eosin stain. These will show vascular tissues and the neurones and excessive glia in the olives, pons, cord, dentate nuclei and cerebellar cortex, and if the Bergmann glia and basket cells are swollen, then these, too, will be shown. Thicker paraffin sections stained by Weil's technique will reveal myelin trouble and sclerosis after a couple of hours' work, especially if a light counterstaining with Van Gieson's stain is carried out. Similar paraffin sections treated with Weigert's mordants 1 and 2 and then with a solution of 0·25% potassium permanganate and 1% oxalic acid (or simply soaked in 1% lead chloride for fifteen minutes) and stained overnight in Mallory's phosphotungstic hæmatoxylin, will reveal the same conditions as well as perivascular gliosis, glial cells and *Gitterzellen*, and the vascular walls. Special glial impregnations like gold sublimate or Holzer's stain show up Bergmann's radiating gliosis, and a microglial stain may be necessary for the shrubberies. These will distinguish between isomorphous and anisomorphous glial arangements. Von Braunmühl's almost foolproof incomparable silver impregnation for argentophile plaques and mesenchymal fibres may be necessary, and Marchi-Busch osmic acid or some other lipoid method may reveal a recent but most important degeneration otherwise perhaps quite overlooked. Cajal's frozen section silver method has been mentioned for the baskets and ascending axones and collaterals. Recently my attention has been drawn to a method whereby thin frozen sections from 10% formalin solution after washing in distilled water may be placed on a slide, blotted and then covered for five minutes in a solution of 1% thionin blue in 0·5% tartaric acid. A coverslip is then applied and the

excess of stain removed and the edges are treated with varnish. Neurones, myelin fibres and the vascular system of the cerebellum at once allow of an excellent examination. A similar treatment for paraffin section produces a useful "Nissl" effect.

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PHYSIOLOGY OF THE CEREBELLUM.[1]

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ALTHOUGH physiologists have long recognized the general nature of the part played by the cerebellum—namely, the maintenance of equilibrium and the regulation of postural tone in accordance with the requirements of muscular movement—they have only just begun to understand the inner mechanism of this great mass of complex neurones. Recent investigations, especially those of Larsell, Dow and Fulton, have provided much new information, and enabled satisfactory interpretation to be made of many previously established facts. Three methods of approach have been employed in the investigation of cerebellar function, namely, ablation experiments, stimulation experiments and action current studies. In the present paper, the results of these different investigations will be considered for each subdivision of the cerebellum. The well-known phylogenetic classification into neocerebellum and palæocerebellum will be adopted in the following description: from the functional point of view, this system is preferable to any of the purely morphological systems of subdivision, because the boundaries between areas of different functional significance are not necessarily anatomical landmarks.

The Effects of Total Removal of the Cerebellum.

The earliest recorded experiments on cerebellar ablation were performed, according to Fulton,[(1)] in 1664 by Thomas Willis, who suggested that the cerebellum controlled involuntary activities, because the heart stopped when this part of the nervous system was manipulated, and the animal died if it was removed. The foundations of modern cerebellar physiology were not laid until 1809, when Rolando described ablation experiments on several animal species, and noticed disturbances of voluntary movement without sensory or mental changes. These findings were confirmed in 1824 by Flourens, who described many of the now familiar symptoms of cerebellar deficiency. He was struck by the similarity between the signs of cerebellar injury and acute alcoholism, and on the strength of this observation, argued naïvely that alcohol probably acted on the cerebellum. The effects of total cerebellar ablation were first studied in detail by Luciani towards the end of last century. He divided the recovery period after total cerebellectomy in dogs in three stages, which he called the periods of "functional exaltation", "deficiency phenomena" and "compensation". Although these stages are not strictly distinct, they are still used as a basis for describing the results of cerebellectomy in animals. During the period of "functional exaltation", tonic spasm of the antigravity muscles is present, as in decerebrate rigidity, with periodic exacerbations of this spasm accompanied by head retraction and opisthotonos. The stage of "deficiency phenomena" begins after a varying number of days, when the animal first attempts voluntary movements, and is characterized by muscle weakness, hypotonia and tremor with incoordination of movement, referred to by Luciani as "asthenia", "atonia" and "astasia", the well known, but not strictly accurate, "Luciani triad". The third stage, that of compensation, begins after a few weeks and may continue for two years or longer. During this period, the signs become progressively less marked and the animal's motor capacity undergoes gradual improvement. More recent investigations have confirmed most of Luciani's findings, and have greatly amplified them (especially in the case of primates), thereby revealing new facts which help to explain some of the discrepancies between the clinical signs of cerebellar disease and the results of animal experiments. It is illuminating to compare the effects of cerebellectomy in lower mammals and in primates. After this operation in the cat and dog, the period of extensor rigidity with seizures of opisthotonos is very pronounced, and may last for as long as a week or ten days. The whole picture suggests a hyperactivity of tonic antigravity reflexes, a viewpoint which is further strengthened by the observation that removal of the cerebellum in a decerebrate animal increases the existing extensor rigidity (Sherrington,[(2)] Pollock and Davis[(3)]). Tremor and ataxia do not appear until the animal attempts voluntary movements (usually after three or four days), and the typical cerebellar picture is not developed for about two weeks. Hypotonia is not a constant feature of the fully developed syndrome, and it has been shown

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association on July 22, 1940.

by de Barenne that it occurs only when the vestibular nuclei have been damaged during the cerebellar ablation. In fact, these animals continue to show a tendency to over-action of the extensor postural mechanism, as indicated by a persistent increase in the positive supporting reaction (Magnus[4]). In monkeys, on the other hand, complete cerebellectomy always produces definite and lasting hypotonia. The release from inhibition of tonic postural reflexes is much less obvious in these animals, and may consist merely in exaggeration of the positive supporting reaction (Fulton[3]). Decerebellate monkeys attempt voluntary movements within twenty-four hours of the operation, and within forty-eight hours pronounced ataxia and tremor develop (Fulton,[5] Aring and Fulton[2]). Hypotonia appears at this time and remains indefinitely. In the higher apes, ataxia and hypotonia are even more pronounced than in monkeys (Botterell and Fulton[7]). The effects of cerebellar deficiency in man have been exhaustively investigated by Gordon Holmes,[8] who had unique opportunities for studying cases of gunshot wound of the head during the war of 1914-1918. According to him, extensive destruction of the human cerebellum is followed within a few hours by definite muscle hypotonia, which increases progressively for a week or ten days after the injury. Besides hypotonia, there appear tremor, muscle weakness and fatigability, and all the well-known signs of failure of integration between different muscle groups known collectively as asynergia. True nystagmus and symptoms of giddiness and rotation usually do not occur, and there is rarely any indication of exaggerated postural reflexes.

This comparison of the effects of complete cerebellar ablation in different species shows that, while release from inhibition of postural reflexes is a characteristic sign of cerebellar deficiency in lower forms, muscle hypotonia becomes more and more obvious as we ascend the evolutionary scale, with diminishing evidence of exaggerated tonic reflexes, until in man diminished tonus is the predominant finding. Indeed, Walshe[9] goes so far as to state that in man "all the component symptoms of cerebellar ataxy are nothing more than manifestations of loss or impairment of muscle tone". What, then, is the explanation of this difference in the effect of cerebellar removal in animals of different evolutionary complexity? Without doubt, it is connected with the progressive growth of the cerebral cortex and associated neocerebellar structures, so that, in primates, the primitive postural patterns necessary to lower mammals become overshadowed by the greatly expanded motor system required for their increased range of voluntary movement. The functions of the two great phylogenetic subdivisions of the cerebellum must therefore be considered separately.

Functions of the Palæocerebellum.

The boundaries of the palæocerebellum have not been finally settled, as it is not a distinct anatomical entity. According to Larsell,[10] it comprises the anterior lobe (lingula, *lobulus centralis* and culmen[1]), the pyramis, paraflocculus and uvula, and the flocculo-nodular lobe, together with the *nucleus fastigii*, most of the *nucleus interpositus* and the dorso-medial portion of the anterior half of the dentate nucleus. The connexions of this portion of the cerebellum are primarily spinal and vestibular; this fact suggests that its function is the integration of low-level reflex activities such as effect suitable adjustments of posture in response to bodily movement. The ventral spino-cerebellar tracts terminate chiefly in the anterior lobe, but some fibres end in the *lobulus simplex* of the neocerebellum. The dorsal spino-cerebellar tracts pass to the pyramis, uvula and paraflocculi; fibres from brain-stem nuclei such as the trigeminal probably end here too. Fibres from the vestibular nuclei pass chiefly to the flocculo-nodular lobe, although a few end in the lingula and uvula (Ingvar[14]). Direct vestibular fibres also reach the flocculo-nodular lobe and the fastigial nuclei (Dow[13]). Some fibres from the pontine nuclei and the region of the inferior olives have been shown to establish connexion with almost every part of the palæocerebellum (Dow[13]). The efferent connexions of the palæocerebellum are effected chiefly through the medial group of central nuclei; but Dow has shown that the flocculo-nodular lobe projects directly to the vestibular nuclei (Figure I). The *nucleus fastigii* receives fibres from the entire palæocerebellum, and projects to the opposite vestibular nuclei

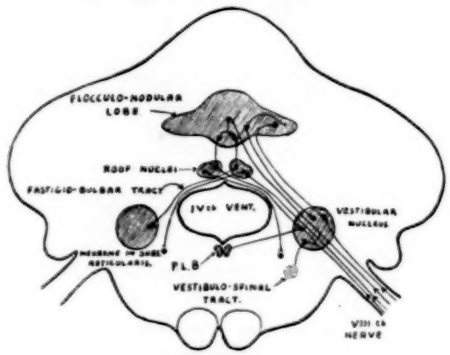

FIGURE I.

and reticular substance by means of the fastigio-bulbar "tract", which consists of the majority of the juxta-restiform or periventricular group of fibres together with Russell's hook bundle (Ferraro and Barrera[15]). The *nucleus interpositus*, consisting of the globose and emboliform nuclei, is palæocerebellar except for a few cells of the *nucleus emboliformis*, and receives fibres from the entire palæocerebellum. It projects entirely to the large-celled portion of the red nucleus via the superior peduncle (Figure II).

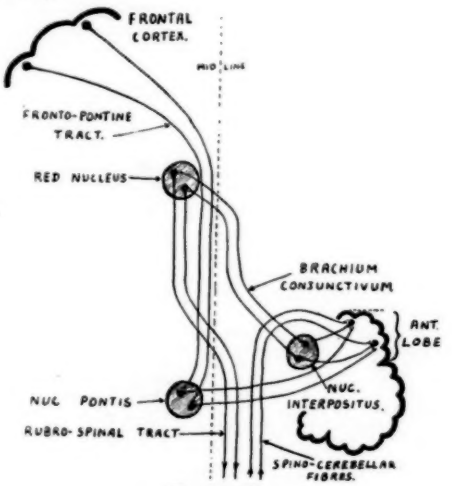

FIGURE II.

The Flocculo-Nodular Lobe.

Isolated ablation of the flocculo-nodular lobe, the chief vestibular region of the cerebellum, has been carried out in primates by Dow.[17] As would be expected, such an animal shows pronounced disturbance of equilibrium, without any abnormality of reflexes or disturbances of voluntary movement. Removal of the uvula as well aggravates the signs, but isolated destruction of any other part of the cerebellum fails to produce disturbances of balance. In the higher apes, isolated destruction of the

[1] Most authorities consider the culmen as palæocerebellar (Larsell,[10] Larsell and Dow,[11] Fulton[3]); there is, however, suggestive evidence that it is associated with cortico-ponto-cerebellar fibres of recent phylogenetic origin (Abbie[12]). Pontine fibres certainly reach the culmen (Dow[13]); but until these are proved to belong to recently acquired cortical projections, the question is best left open.

nodulus causes great interference with balance, with oscillation of the head and neck and so-called "trunk ataxia", frequent falling and abducted gait, the syndrome being almost identical with that produced in children by mid-line cerebellar tumours, which often arise directly from the nodulus. Ferraro and Barrera[16] describe similar disturbances in monkeys after the production of lesions of the roof nuclei and fastigio-bulbar tracts. Stimulation by faradic currents of the flocculo-nodular lobe, when properly controlled to avoid spread of the stimulus, has not yielded constant results up to date. Study of the electrical activity of this portion of the cerebellum has provided final proof that its function is vestibular. Dow[13] has shown that stimulation of the eighth nerve produces a distinct electrical response in the flocculo-nodular lobe; some response was also detectable in the uvula, lingula and fastigial nuclei. Physiological confirmation has thus been obtained of conclusions drawn from anatomical investigations.

The Anterior Lobe.

In lower mammals and birds, removal of the anterior lobe of the cerebellum causes pronounced release of postural mechanisms, with increase of extensor tonus and exaggeration of stretch reflexes. In primates this operation produces much less conspicuous effects, although Dow (see Fulton[1]) has demonstrated an exaggeration of the positive supporting reaction in the monkey after ablation of this lobe. It is practically certain that the great increase in postural reflexes seen in lower forms after total cerebellectomy is due to interruption of the palæocerebellar projections of the anterior lobe. Stimulation of the anterior lobe inhibits decerebrate rigidity in nearly all vertebrates, this being most easily shown in birds. In the intact animal, stimulation of this lobe will inhibit extensor postures, and on withdrawal of the stimulus there is a pronounced "rebound" contraction of the extensors. These phenomena were first observed by Sherrington,[18] and have since been confirmed by Miller and Banting[19] and many other workers. Thus the results of ablation and of stimulation of the anterior lobe indicate that its normal function is the restraint of primitive extensor postural patterns, through its projections to the red nucleus and possibly to other brain-stem centres. This conclusion is supported by the recent work of Dow,[20] who finds that the electrical activity of the anterior lobe varies inversely with the degree of tonus in the extensor muscles.

The Uvula.

The function of the uvula, as we have seen, is partly vestibular; its removal causes transient disturbances of equilibrium. Since it receives only a scattering of vestibular fibres, and has spinal and bulbar connexions, it probably performs other functions, but these have not yet been disclosed.

The Paraflocculi.

Removal of the paraflocculi does not give rise to any conspicuous disturbance, and no specific response to stimulation has been described. Electrical responses are set up in this portion of the palæocerebellum when the olivary nuclei and pons are stimulated, but no real light has yet been thrown on its functional significance.

The Pyramis.

Isolated removal of the pyramis does not disturb balance or reflex action; but Dow[17] has described a curious finding in monkeys after this operation had been performed. These animals appeared to be unable to prevent themselves from crashing into clearly visible obstructions. Since faradic stimulation of the pyramis is known to produce eye movements, Dow has suggested that this defect may be due to impairment of the capacity to judge distance, resulting from faulty integration of proprioceptive impulses from the eye muscles.

Summary.

The functions of the palæocerebellum may therefore be summarized as follows:

1. The vestibular portion, consisting of the flocculo-nodular lobe, the uvula, the lingula and the associated portions of the fastigial nuclei with their medullary projections, may be considered as extramedullary extensions of the vestibular nuclei, and subserve similar functions. They are essential for the proper coordination of labyrinthine information with muscular movement in the intact animal, but play no part in the primitive labyrinthine reflexes demonstrated by Magnus and de Kleyn in the decerebrate preparation.

2. The remainder of the palæocerebellum is primarily concerned with the restraint of excessive activity of the postural reflexes, and produces appropriate changes in postural pattern in accordance with the requirements of muscular movement as signalled by the spino-cerebellar and bulbo-cerebellar tracts. In addition, the pyramis may be concerned with the capacity to judge distances by integrating proprioceptive impulses from the eye muscles.

Functions of the Neocerebellum.

The neocerebellum, the most newly acquired, and in primates the most important part of the cerebellum, consists of that portion of the cerebellar cortex lying between the *fissura prima* and the *fissura prepyramidalis* (see footnote on page 168), together with the major part of the dentate nucleus, a few cells of the emboliform nucleus and associated fibre tracts. The afferent connexions are derived chiefly from the pontine nuclei, in which terminate the cortico-pontine tracts, so that each half of the neocerebellar cortex is under the influence of the opposite cerebral hemisphere. Fibres from the opposite inferior olive (neoolivary portion) also reach the neocerebellar cortex (Holmes and Stewart,[21] Dow[13]), and a few fibres from the ventral spino-cerebellar tract end in the anterior part (the *lobulus simplex*) (Larsell[10]). According to McNalty and Horsley,[22] the neo-cerebellar cortex receives fibres from both spino-cerebellar tracts; but this is denied by Larsell, whose studies have shown that the neocerebellar afferents are predominantly cortico-pontine. The neocerebellar Purkinje cells project to the posterior half and ventro-lateral portion of the anterior half of the dentate nucleus as well as to part of the emboliform nucleus (Fulton[1]). Some of these Purkinje axones may pass directly into the superior peduncles without any synaptic interruption (Sachs and Fincher[23]), but the majority terminate in cells of the *nucleus dentatus*. The axones of these cells pass through the superior peduncles to reach the opposite red nucleus. Here, according to Mussen,[24] about half the fibres end in the small-celled portion, and a few in the large-celled part, while the remainder pass on to end in the latero-ventral nucleus of the thalamus. A fresh relay of fibres connects this region with areas IV and VI of the cerebral cortex (Walker[25]). This pathway from the dentate nucleus to the cerebral cortex is now considered to be the chief efferent connexion of the primate neocerebellum.

Ablation Experiments.

Removal of the neocerebellar cortex produces very slight and transient effects in lower mammals. In monkeys, however, this operation is followed by pronounced hypotonia and impairment of skilled movements; in higher apes the same disturbances are seen, but are of greater severity (Botterell and Fulton[7]). Unilateral ablation of the neocerebellar cortex in primates causes ipsilateral hypotonia and clumsiness with some disturbance of gait, the signs lasting for several weeks. Bilateral removal produces similar but more severe disturbances in all four limbs, together with gross abnormalities of gait. Tremor appears only if the dentate nucleus has been injured. Interference with this nucleus greatly impairs the animal's power of recovery from the effects of the cortical lesion. Small ablations of any portion of the neocerebellar cortex in chimpanzees produce hypotonia *et cetera* in both ipsilateral limbs; hence Botterell and Fulton conclude that there is no evidence of discrete spatial localization of muscle groups within the cerebellar cortex. Similar findings in man have been reported by Gordon Holmes.[26]

Stimulation Experiments.

Stimulation of the neocerebellar cortex has been carried out by various investigators with widely differing results. Ferrier[26] obtained inconstant responses of the head and eyes to faradic stimulation of the cerebellar cortex. Horsley and Clarke[27] came to the conclusion that such stimuli were almost without effect on the cerebellar cortex, but that responses were readily obtainable from stimulation of the central nuclei. On the other hand, Mussen[24] claimed to have produced movements of almost every muscle in the body by faradic stimulation of different areas of the cerebellar cortex, and published an elaborate scheme of the cortical localization of these responses. These findings have not been confirmed, and cannot be accepted in view of the recently acquired knowledge of the functional significance of the different cerebellar subdivisions. Mussen's results must be regarded as having been caused by spread of current to neighbouring, more excitable structures. The modern viewpoint on the question of neocerebellar excitability is expressed by Fulton,[1] who states that "faradic stimulation of the neocerebellum, except in transitional zones such as the *lobulus simplex*, is entirely without effect on the skeletal musculature". Anatomical considerations alone show that the neocerebellum can affect skeletal muscles only indirectly through the cerebral cortex, since it projects almost exclusively to the motor and premotor areas and to the small-celled part of the red nucleus, which has few spinal connexions. Reference will be made later to the nature of the cerebellar influence on the cerebral cortex. Attempts have been made to determine the excitability of the central nuclei by a number of investigators. Stimulation of these nuclei has been carried out by Horsley and Clarke,[27] Sachs and Fincher,[25] Magoun, Hare and Ranson,[28][29] Miller and Laughton,[30] and the results are in general agreement. The dentate nucleus has a higher threshold than the other nuclei; but strong stimulation may produce increased flexor tone in the ipsilateral limbs, with inhibition of extensors, or else alternate flexion and extension of the forelimb. The general tendency is for postural patterns to appear slowly during prolonged stimulation, followed by a "rebound" phase when the stimulus is withdrawn, in which the reverse of the original posture is held for some time. It is impossible to say whether the neocerebellar components of the central nuclei are concerned at all in these responses, for it is obvious that simultaneous excitation of palæocerebellar neurones is unavoidable when the dentate nucleus is stimulated.

The spontaneous electrical activity of the neocerebellar cortex is essentially the same as that found in other cortical areas, an observation which is not surprising in view of the known histological uniformity of the cerebellar cortex. The electrocerebellogram consists of groups of small, rapid waves occurring at a rate of 100 to 200 per second, the amplitude being about 20 microvolts (Dow[20]). Spiegel[21] has shown that this spontaneous activity is reduced, but not abolished, by section of all the afferent tracts to the cerebellum. Dow[12] has studied the changes in the cerebellar electrogram produced by stimulation of the different afferent pathways, and his results provide striking confirmation of the conclusions drawn from degeneration experiments as to the termination of these tracts. Reference has already been made to these oscillographic findings in connexion with the palæocerebellum; Dow also demonstrated that responses were set up in the neocerebellar cortex only when the regions of the pons and inferior olives were stimulated. No reports have yet appeared of the effects on the cerebellar electrogram of stimulation or removal of areas of the cerebral cortex. The cortico-ponto-cerebellar pathways must have some specific effect on cerebellar activity, and one can confidently predict that the oscillographic method will provide much useful information on this point before long.

Summary.

To summarize, experiment has shown that the neocerebellar cortex, though not of great importance to lower mammals, is essential in primates for the maintenance of muscle tone and the proper execution of skilled movements; that the dentate nucleus can exert by itself enough control over voluntary movements to prevent the development of tremor and hasten the process of recovery from cortical ablation; that disturbances of equilibrium and posture (other than those due to hypotonia) are not produced by neocerebellar injury; and that there is no discrete representation of the skeletal musculature in the cerebellum. The problem which now confronts us is the exact nature of the close interaction between the cerebrum and neocerebellum, which ensures in primates such amazing efficiency of intricate voluntary movements.

Interrelation of Cerebrum and Cerebellum.

Clinicians have long recognized the importance of the cerebral cortex in the manifestations of cerebellar lesions. The late James Collier is quoted by Fulton[5] as having said: "It is very easy to recognize a cerebellar lesion, because the patient walks with his cerebral hemispheres." The physiological basis for this piece of clinical intuition has now been provided, largely by the work of Fulton. Consideration of the effects of neocerebellar ablation show that the typical signs of asynergia appear only when voluntary movement is attempted. This observation led Fulton to investigate the effect on cerebellar tremor of ablation of the cerebral hemispheres. He showed[5] that in decerebrate cats, removal of one cerebral hemisphere profoundly diminished the cerebellar signs on the opposite side. Tremor completely disappeared until late in the recovery stage, when slight tremor recurred, due to innervation from the ipsilateral hemisphere. Removal of both cerebral hemispheres from decerebellate cats caused opisthotonoid seizures and extensor rigidity for a few days, after which the animals survived for about two weeks without seizures or rigidity. They carried out vigorous spontaneous movements, and performed reflexes, such as the scratch reflex, without any sign of ataxia or tremor. Thus direct proof was obtained of the cerebral origin of decerebellate tremor and asynergia.

Aring and Fulton[6] have carried out extensive studies of the relation between the primate cerebellum and the motor regions of the cerebral cortex. It will be recalled that there are two chief motor systems in primates—namely, the pyramidal system and the extrapyramidal system. The pyramidal system consists of the giant Betz cells and their axones which form the pyramidal tracts. These cells occur in the fifth layer of area IV, the classical "motor area", which yields discrete responses of individual muscles on electrical stimulation. Ablation of area IV or section of the pyramidal tracts causes flaccid paralysis. The extrapyramidal system consists of widely spread cortical motor neurones which project to the basal ganglia, the *substantia nigra*, the red nucleus, the pontine nuclei and possibly directly to the spinal motor neurones. These extrapyramidal neurones are found in area IV, area VI (the "premotor" area), area VIII (the frontal eye field), areas I, II, III and V of the parietal lobe, and area XXII of the temporal lobe. Stimulation of these areas in the absence of the pyramidal system produces either slow, complex and apparently purposeful movements of eyes, head, trunk and limbs, or else causes inhibition of some existing postural pattern, especially extensor postures. Removal of extrapyramidal cortical areas tends to cause release of postural patterns and loss of skilled movements. Thus ablation of area VI produces pronounced spasticity and increase of tendon reflexes, with forced grasping and loss of previously acquired patterns of skilled movement. It has already been pointed out that the chief projections of the neocerebellum terminate in areas IV and VI of the cerebral cortex. Working with decerebellate monkeys and chimpanzees, Aring and Fulton found that: (i) ablation of area IV diminished, but did not abolish, cerebellar tremor; (ii) bilateral ablation of areas IV and VI abolished cerebellar tremor as completely as removal of both cerebral hemispheres in the cat; (iii) ablation of area VI alone caused augmentation of all the signs of cerebellar deficiency.

Thus cerebellar tremor and ataxia arise when areas IV and VI are working without cerebellar control, and area VI

contains the neurones chiefly responsible for cerebral compensation of cerebellar deficiency.

Two other facts have been brought to light, which are helpful in revealing the nature of the cerebellar influence on cortical activity. The absence of obvious effects on the skeletal musculature of neocerebellar stimulation has already been noted; but Rossi (see Fulton[131]) has described a lowering of the threshold to stimulation of area IV during faradic excitation of the contralateral neocerebellar cortex, and as a corollary to this, Walker[133] has demonstrated a distinct increase in the amplitude and frequency of the spontaneous electrical rhythm of area IV, and to a lesser degree of area VI, during stimulation of the opposite neocerebellar cortex. A similar effect has recently been noted in our own laboratory. Adrian and Moruzzi[135] have shown that even in the absence of voluntary movement the pyramidal axones are continually discharging streams of impulses which vary in accordance with the degree of electrical activity in the motor cortex. These impulses serve to maintain tone in the voluntary muscles, because when they are cut off by section of the pyramids, flaccid paralysis results (Tower[134]). It seems probable that the neocerebellum, which is known to possess considerable spontaneous activity, at all times sends impulses to areas IV and VI, thereby keeping these regions in a state of moderate excitation even when the animal is at rest. Thus muscle tone is preserved, and a smoothing background of activity is provided, on which are grafted the changes that produce voluntary movements. The hypotonia which follows neocerebellar lesions is thus due to diminished activity of the pyramidal neurones after removal of this tonic cerebellar excitation (Figure III).

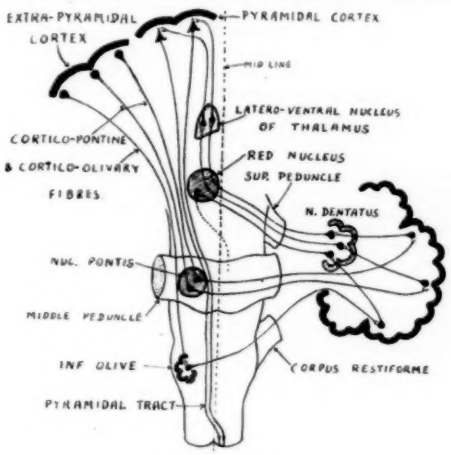

FIGURE III.

The nature of the reciprocal effect of the cerebral cortex on cerebellar activity is much more obscure. Even the exact origin and distribution of the cortico-cerebellar activity is much more obscure. Even the exact origin and distribution of the cortico-cerebellar pathways are not clearly understood. Fronto-pontine tracts are known to occur in most mammals, and in primates probably arise chiefly in area VI, but partly in area IV. According to Levin,[85] some fibres may also arise in the frontal association areas (areas IX, X, XI and XII). Parieto-pontine and temporo-pontine fibres are well developed in primates, but are absent or insignificant in many lower forms. The fronto-pontine fibres descend to the pontine nuclei in the medial one-third of the *crus cerebri*, while the other cortico-pontine tracts lie in the lateral region of the crus. Cortico-olivary fibres arise from the region of the inferior retro-central sulcus (Mettler[150]), and form another possible descending pathway from cortex to cerebellum. The distribution to the cerebellum of these descending tracts is not yet clear, but it is certain that the majority of the cortico-pontine fibres, after relaying in the pontine nuclei, end in the contralateral neocerebellar cortex (Figure III). However, the oscillographic studies of Dow[123] have shown that some pontine fibres pass to palæocerebellar regions (pyramis, paraflocculus and culmen), as well as to the neocerebellar cortex; and the evidence brought forward by Abbie,[122] based on comparative neurology, that the culmen is associated with temporo-pontine projections, indicates that some revision may be necessary of our conception of the distinction between neocerebellar and palæocerebellar structures. It is likely that considerable overlapping of function takes place in transition zones, so that a region like the culmen may consist of neurones from both systems. Indeed, Dow noted that in some cases stimulation of two different afferent systems (pons and spino-cerebellar tracts) evoked responses in the same region of the cerebellum; furthermore, the electrical waves produced by these two systems often differed in sign and shape. Dow offers the interesting suggestion that different afferent fibre systems—namely, the "mossy" and "climbing" fibres—may be involved in the two cases. There is obviously a very promising field here for future investigation.

The exact part played by these cortico-cerebellar pathways in controlling cerebellar activity is not clear. In lower mammals it has been shown that they are important factors in the regulation of posture. In the cat, Warner and Olmsted[127] demonstrated that removal of the frontal cortex caused contralateral extensor rigidity; stimulation of the cut surface inhibited this rigidity, but failed to do so after section of the middle cerebellar peduncle on the rigid side. It is well established that stimulation of the anterior lobe of the cerebellum inhibits extensor rigidity (Miller and Banting[139]), and the striking increase in extensor tone after cerebellar ablation in the cat has already been noted. The function of the fronto-ponto-cerebellar pathway in lower mammals therefore seems clear; it exerts a tonic action on the anterior lobe of the cerebellum, which in turn inhibits, via the red nucleus and rubro-spinal tracts and probably by other pathways, the primitive extensor posture subserved by the vestibular nuclei and vestibulo-spinal tracts (Figure II). In primates, removal of area VI of the frontal cortex releases a postural pattern closely akin to decerebrate rigidity, but the part played by the cerebellum is obscure. The mechanism cannot be the same as in the cat, because removal of the primate cerebellum is not followed by spasticity, but by hypotonia. Perhaps in primates the same mechanism is present in rudimentary form, but is overshadowed by the vastly more important neocerebellar-pyramidal system, interference with which causes loss of muscle tone. This conjecture is supported by the occasional occurrence in humans of spasticity with partial lesions of the superior peduncle (Holmes[85]). The pathways through which the primate frontal cortex inhibits excessive extensor tonus must be largely extracerebellar, and are probably associated with the basal ganglia and *substantia nigra* systems or by direct projections to the anterior horn cells.

The role of the more recently acquired cortico-cerebellar pathways is infinitely more complex than that of the fronto-pontine tracts. They have developed *pari passu* with the acquirement of increasing skill and complexity of voluntary movement, and bring the neocerebellar cortex into intimate association with the higher association areas of the brain. Thus when a movement is willed, instructions reach the neocerebellar cortex as well as the cerebral motor neurones; these can therefore be governed and their responses restrained and smoothed by appropriate cerebellar activity. In the absence of these pathways the cerebellum would work blindly and would be unable to exert its controlling influence on voluntary movements. The production of cerebellar signs by certain supratentorial lesions in man may be due to interference with these important tracts or their cells of origin.

Conclusion.

It can be seen from this survey of experimental work on the cerebellum that understanding is slowly dawning of the way in which this intricate organ performs its

duties. Recognition of the differences between the cerebellum of primates and of lower forms has helped to bridge the gap, conspicuous for so long, between the signs of cerebellar lesions in man and the results of experiments on animals. Further work must be devoted to such questions as the exact origin and distribution of the cortico-ponto-cerebellar pathways and the nature of their normal activity; the details of the tonic influence exerted by the neocerebellum on the cerebral cortex; the functions of such areas as the paraflocculus and pyramis. The oscillographic method of studying neural activity will certainly play a large part in future investigations of the cerebellum, and will help us to understand still more of its extraordinarily complex activity in maintaining bodily equilibrium, adjusting posture to the needs of the moment and ensuring that amazing efficiency of skilled movement which has put the material universe at the disposal of man.

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THE DIAGNOSIS OF CEREBELLAR DISEASE.[1]

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IN so far as this paper is a discussion on tumour of the cerebellum, it is clear that it will exclude from the long list of mimicking disorders the cerebellar degenerations, familial ataxias and polysclerosis which are not characterized by an increase in intracranial tension. Further, it should be borne in mind that there exists in all cases of cerebellar tumour a dual syndrome, a syndrome which presents not only the signs and symptoms of specific lesion of the cerebellar nuclei or cerebello-cortical pathways, but also the clinical manifestations of intracranial hypertension. The latter component is known locally to some as the "H.V.P." syndrome (the letters standing for headache, vomiting and papilloedema), and is such a prominent feature in all the disorders paraded for differential diagnosis that it requires further elaboration.

Intracranial Hypertension.

The symptoms of intracranial hypertension can be enumerated in the following way; but one must be mindful of the fact that they tend to be fugitive in the early stages (mid-line cerebellar tumours in particular demonstrate this periodicity).

1. Increased pressure betrays itself early by headache, the severity of which in many instances depends on the degree of closure of the cranial sutures. The location of pain is usually frontal; but when it occurs in the occipital region together with nuchal stiffness it is very suggestive of a posterior lesion, a lesion which nevertheless may be in the nature of tonsillar herniation and may occur as a late feature in any type of hydrocephalus. The typical bursting, throbbing ache is exaggerated by straining at stool, coughing, stooping (in one instance by trotting on horseback), and it was Cushing who first recorded the observation that children with mid-line astrocytomata were usually of a placid nature and became ward pets, no doubt owing to avoidance of the tears and tantrums which aggravated their headache.

2. Unheralded projectile vomiting, usually dissociated from nausea, is the classical accompaniment of headache.

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neuro-Surgery of the New South Wales Branch of the British Medical Association on September 19, 1940.

The early morning occurrence falls into line with the early morning onset of "hypertensive" headache, which seldom fails to follow three to four hours' sleep in the horizontal position, a posture which allows of increased vascular engorgement and local œdema.

3. Disturbances of vision are common; they take the form of either transitory sixth nerve palsies or impairment of visual acuity, and are often first observed at school when a child screws up one eye to avoid double images or experiences difficulty in reading blackboard figures. Harvey Cushing, in 1931, lamented the fact that as a result of late diagnosis, a quarter of the children in his series of cases suffering from cerebellar astrocytoma were preamaurotic. He did not refer to the intermittent amblyopias occurring with bouts of headache, but to the effects of secondary optic atrophy, the aftermath of undiagnosed papillœdema.

4. Of the whole symptom complex, the dramatic occurrence of "convulsions" is the most terrifying for relatives or parents, and calls for immediate medical consultation. These seizures not only range from giddy attacks to syncope with pallor and stiffening, and to the classical opisthotonic cerebellar fits, but also take the form of typical epileptiform tonic-clonic seizures. The attacks are precipitated on a background of raised intracranial pressure by any straining which momentarily sends a back pressure along the intracranial jugular tributaries. This sudden venous engorgement increases the pressure within the cranium to blood systolic level; the ensuing syncope or convulsion being an index of the degree of cerebral ischæmia so produced.

Webster and Weinberger consider that the "fits" associated with tumours of the cerebellum are of this nature—that is, they are a physiological decortication rather than a mechanical decerebration by direct pressure on the mid-brain. But, whether one or both of these factors operate in the late stages of posterior fossa tumours, tonic or tonic-clonic fits should always carry graver prognostic valuation than attacks of syncope, which can occur relatively early in hydrocephalus whatever its ætiological factor. (The publication by Penfield of his observations on the production of fits by posterior fossa tumours is awaited with interest.)

5. Late symptoms are concurrent with a more pronounced degree of internal hydrocephalus; hypothalamic disturbances such as drowsiness, adiposity and polyuria are an index of dilatation of the third ventricle; loss of concentration and memory for recent events, and facetiousness indicate cortical atrophy and resemble the emotional changes and *Witzelsucht* of frontal lobe tumours and of disseminated sclerosis; whilst dysequilibrium is a measure of tonsillar herniation.

Added to these are the definite signs of raised intracranial pressure.

6. Enlargement of the head in children may or may not pass unnoticed, and indeed so great is the normal variation that knowledge of head circumference according to age is of little value. Progressive measurements may be more helpful if compared with the average rate of growth *per annum*; increments of 10 centimetres in the circumference are expected between the age periods of three to five years, five to eight years, and eight to thirteen years respectively. On percussion these skulls with separated sutures emit the classical "cracked-pot" sound of MacEwen.

7. Bilateral papillœdema is invariably present, but the early and late stages are difficult to determine, and may pass unnoticed in non-cooperative children, whose restlessness permits only a fleeting glimpse of the fundus. Hence examination of these patients is most successfully made under basal anæsthesia with "Avertin" ("Bromethol"), with or without conjunctival instillation of homatropine. Unilateral papillœdema has no localizing value in lesions below the tentorium.

8. The lumbar puncture pressure is consistently raised above 160 millimetres of cerebro-spinal fluid—a figure taken empirically as the upper limit of normality.

9. There is lastly the radiological evidence of intracranial hypertension, separation of sutures, digital markings or thumbing, an appearance which has to be carefully assessed, as it may normally be present to a slight degree up to the age of twenty-five years and which is due to compression and disappearance of diploic tissue over the summits of the gyri, and ballooning of the sella, an effect produced by the forward and downward pulsating pressure of the dilated third ventricle. To these may be added the thinning and bulging of the occipital bone, so frequently observed in cases of posterior fossa tumour.

Thus are presented briefly the oustanding clinical features of the "H.V.P." syndrome, features which are common to all the following conditions listed for discussion, although the prominence of cerebellar signs and symptoms in certain cases requires the subdivision of cerebellar and extracerebellar lesions into two groups.

Schema for Differential Diagnosis.

The following is a schema for the differential diagnosis:

1. Primary cerebellar lesions:
   - (*a*) Signs and symptoms of raised intracranial tension predominating.
     - (i) Mid-line and small hemisphere tumours not involving nuclei.
   - (*b*) Cerebellar signs and symptoms predominating.
     - (ii) Mid-line and hemisphere tumours involving nuclei.
2. Extracerebellar lesions:
   - (*a*) Signs and symptoms of raised intracranial tension predominating:
     - (i) Toxic hydrocephalus
     - (ii) Basal arachnoiditis.
     - (iii) Lead encephalopathy.
     - (iv) Third ventricle tumours.
     - (v) Pineal tumours.
     - (vi) Miscellaneous group.
   - (*b*) Cerebellar signs and symptoms predominating and confusing the issue:
     - (vii) Infratentorial tumours, eighth nerve tumour for example.
     - (viii) Supratentorial tumours, chiefly frontal.

It may be asked whether disseminated sclerosis can be lightly dismissed on the grounds of normality of intracranial pressure. Superficially, no. This is because of the confusion that may arise in the rare cases (4·8%) of the remitting unilateral cerebellar type, especially if they are characterized by a papillitis greater than two diopters. Abnormal cytology and a paretic or leutic curve should aid in the diagnosis, for examination of the cerebro-spinal fluid in cases of cerebellar tumour rarely reveals any other abnormality than a slight but fairly constant rise in protein content.

Primary Cerebellar Lesions.

Primary Cerebellar Lesions in which Signs and Symptoms of Raised Intracranial Pressure Predominate.—The primary cerebellar lesions of the first group embrace the small hemisphere tumours, for example, the astrocytomata and rarer tuberculomata and hydatid cysts which, whilst large enough to cause obstruction to the flow of cerebro-spinal fluid in the aqueduct or bottle-neck of the incisura, are yet not great enough to interfere with the dentate nucleus projection to the cortex. Secondly, by far the larger group is that of the mid-cerebellar subvermis tumours, including: (*a*) the small and large cyst astrocytomata, occurring most commonly at the beginning of the second decade; (*b*) the solid medulloblastomata, which seed by drop metastases into subarachnoid spaces and down the spinal cord, the origin of which is from undifferentiated cells of the nodulus, and the peak incidence of occurrence of which is in the latter half of the first decade; (*c*) the rarer ependymomata of adolescence; (*d*) the still rarer hæmangioblastomata, occurring in adults between the ages of thirty and forty years, and often associated with retinal hæmangiomata and port wine birth-marks on the neck or face.

It is to the mid-line cystic astrocytomata that Harvey Cushing's classical description applies. The pre-adolescent child towards the end of his first decade, possibly after a fall or whooping-cough representing minor cerebral pressure insults, begins to have early morning headache and vomiting. The vomiting may occur whilst he is in bed, or

unexpectedly at the breakfast table. As there is no associated nausea the meal is completed and the child proceeds as usual to school. Subsequent attacks are attributed to gastro-intestinal upsets and are successfully treated by laxatives. There is an insidious increase in the size of the head. Periods of remission become shorter in duration, until it is observed at school that the sight is poor. Then follows the onset of vague cerebellar symptoms, tripping, inability to keep up with the other children at play, fatigability, drowsiness and possibly fainting attacks or tonic convulsions.

On examination the child displays all the signs of intracranial hypertension together with dysequilibrium. This takes the form of an unsteady, cautious gait on a wide base with a tendency to fall in any direction, but usually backwards. To these findings may be added the occasional appearance of scattered ataxias, speech defects and possibly nystagmus.

With regard to the exclusion of disorders listed in the schema for differential diagnosis [particularly the corresponding group in subdivision (*b*)] special investigations (enumerated later) are carried out. In the routine procedures a lumbar puncture is performed, every care being taken with a rubber manometer 18 inches long in the withdrawal of 1·5 to 2·0 cubic centimetres, a quantity of cerebro-spinal fluid found to be sufficient for protein estimation and cell count. Any other procedure failing adequately to control the flow of cerebro-spinal fluid may well precipitate tonsillar impaction and acute medullary compression, characterized by nuchal pain and rigidity and stertorous irregular breathing, which is likely to fail within one to two hours. This condition must always be considered a grave surgical emergency. Artificial respiration and intubation are immediate considerations, and three cubic centimetres of "Coramine" injected intravenously may prove of value as a respiratory stimulant (this was successful in one case encountered by the writer). While instruments are being sterilized, hypertonic solutions are given intravenously in the form of 50 cubic centimetres of 15% sodium chloride solution and 50 cubic centimetres of 50% sucrose solution, and although no immediate effect on the intracranial pressure can be expected, the resultant dehydration occurring after a lapse of twenty to thirty minutes will aid considerably in the reduction of œdema of the impacted parts. However, the only procedure adequate for relief of the herniation and medullary ischæmia is ventricular tapping followed immediately or within a few hours by sub-occipital decompression (removal of the posterior half of the arch of the atlas and of the posterior margin of the *foramen magnum* through a mid-line nuchal incision).

Finally, in all doubtful cases ventriculography is the only certain method of determining the supratentorial or intratentorial position of the lesion. Even then, specific localization in the posterior fossa is often impossible, and to quote E. W. Twining, "we can never say of the fourth with that conviction which we feel about the third ventricle, because the fourth ventricle is not filled, therefore there is an obstructive lesion of the aqueduct; we are sometimes unable to say whether the lesion is in the aqueduct, in the fourth ventricle, in the cerebellum, or in the extracerebellar arachnoid channels".

Primary Cerebellar Lesions in which Cerebellar Signs and Symptoms Predominate.—The second group of primary cerebellar lesions includes the hemisphere lesions of which the astrocytomata form by far the greater percentage and which give rise to unmistakable lateralized cerebellar symptoms dominating the picture of coexisting intracranial hypertension. A history of intermittent headache, unilateral clumsiness or tripping, leads on examination to the unearthing of the well-known complex: (*a*) hypotonia and fatigability, best demonstrated in drooping and tremor of the outstretched hands and the flail knee jerks; (*b*) disorders of voluntary movement, as seen in terminal tremor, inability to perform delicate movements, decomposition, errors in range, rate, direction and force, and difficulty in performing rapidly alternating movements (Babinski's dysdiadokokinesia); (*c*) nystagmus, which, according to the physiologists, is a neocerebellar disorder occurring only when the dentate nucleus is involved (the amplitude of nystagmus is greater on deviation of the eyes to the side of the lesion); (*d*) palæocerebellar vestibular phenomena of past pointing, deviation to the right or left when walking with the eyes closed, and head rotation; (*e*) occasional signs of involvement of the pyramidal tract, or sixth and seventh cranial nerves.

As mentioned previously, cerebellar degenerations and disseminated sclerosis are excluded from the diagnostic field in the presence of a high cerebro-spinal fluid pressure. Eighth nerve tumours can usually be eliminated if a history of early progressive involvement of the eighth, fifth and seventh cranial nerves and late cerebellar involvement is lacking. But contralateral frontal lobe tumours often can be excluded only by brain needling and ventricular air studies. Indeed, it was a fatality in just such a case after a suboccipital exploration which produced negative results that convinced Penfield of the wisdom of routine ventriculography.

With regard to the interpretations of these ventriculograms, Elsberg and Silbert hold that cerebellar hemisphere tumours are associated with a dilatation of the opposite lateral ventricle and a dislocation upwards and forwards of the ipsilateral posterior horn. This has been discredited by the observations of McConnell and Childe on the grounds that the lateral ventricles tend to be dilated and symmetrical; and that so frequently in normal ventriculograms are the posterior horns found to vary in size and shape that abnormalities cannot be taken as diagnostic guides. They further maintain that the only diagnostic point is displacement of the Sylvian aqueduct to the opposite side. The problem is further elucidated by Twining in his exhaustive article "Radiology of the Third and Fourth Ventricles", in which he states how difficult it is to distinguish between artefact-like air locks in anything less than a quarter-inch diameter aqueduct, on the one hand, and actual stenosis by pressure or ependymitis on the other. Nevertheless, he then proceeds to enumerate characteristic, although seldom found, deformities, the spigot-shaped aqueduct of ependymitis, the dilated fourth ventricle of arachnoiditis, the kinked and displaced aqueduct of hemisphere tumours, and the filling defects of subvermis tumours.

Extracerebellar Lesions.

The extracerebellar disorders causing clinical manifestations of intracranial hypertension, with or without vague pyramidal or cerebellar signs, constitute the main diagnostic barrier. A few have been selected for discussion; others, no less important, can be referred to only by name.

Toxic Hydrocephalus.—This condition takes the form of a "hypertensive" hydrocephalus, in which the abundant cerebro-spinal fluid occurring in the subarachnoid spaces, seldom loculated in cisterns or ventricles, is of normal protein content and under greatly increased pressure. In general it occurs as a complication of focal infection situated in the middle ear, the mastoid or the naso-pharynx, or at tooth apices. McAlpine considers the condition to be of an allergic nature, streptococcal toxins causing an over-production of cerebro-spinal fluid by the chorioid plexus. This is an attractive hypothesis, especially as exacerbations are known to occur with recurrent tonsillitis and sinusitis. However, C. P. Symonds considers aseptic thrombophlebitis of the dural sinuses and cerebral veins, with decreased absorption from the arachnoid villi, to be the most likely cause of the specific type complicating middle-ear and mastoid infection; to this he gives the name otitic hydrocephalus.[1] Thus the symptoms may be intermittent, and may coincide with recurrent tonsillitis, sinusitis or periapical infection; or the onset sudden and marked by severe headache, rapid

[1] Recently C. P. Symonds reported three cases of probable thrombophlebitis spreading from the frontal sinuses. In each case pareses or seizures occurred, the lumbar puncture pressure was low, and the cerebro-spinal fluid contained a pronounced increase of cells and protein. All these features distinguish thrombosis from toxic hydrocephalus, in which no localizing signs occur and in which the cerebro-spinal fluid is chemically and cytologically normal.

loss of vision and development of high papilloedema, but with lack of constitutional as well as of focal signs. In resemblance to cerebellar tumour, attacks of syncope and tonic convulsions may occur; but, unlike these posterior fossa lesions, symptoms tend to be most severe at the onset, spontaneous recovery taking place after weeks or months and unrelieved papilloedema subsiding into optic atrophy. Lumbar puncture reveals a pressure usually above 300 millimetres of cerebro-spinal fluid; the fluid, as mentioned previously, is normal as regards protein content and the number of cells. If the presence of a space-occupying lesion in the posterior fossa cannot be excluded, it is wise to demonstrate by ventriculogram and ventricular estimation the presence of small symmetrical ventricles and external hydrocephalus before proceeding with drainage. If, on the other hand, the diagnosis is not in doubt, lumbar puncture drainage can be instituted forthwith, when it will be found possible to relieve the headache and papilloedema by the withdrawal of 30 to 40 cubic centimetres of fluid per day or even twice a week (in amounts sufficient to halve the pressure readings). Ventricular tap and suboccipital decompression and exploration are indicated in those cases only if pressure remains high despite adequate drainage and treatment of the toxic focus.

Basal Archnoditis.—Proliferative changes in the nature of milky opacities and thickening occurring in the leptomeninges, particularly in the basal cisterns and *cisterna magna*, with subsequent blocking of the foramina of Magendie and Luschka, are a sequel to encephalitis and meningitis and indeed to chronic metallic poisoning with lead or arsenic. Dilatation of the third ventricle and iter point to fourth ventricle block, and as it is highly improbable that ventriculography will have been complete enough to indicate the exact nature of the obstruction, most of these patients come to operation and exploration is made through a suboccipital mid-line approach.

Lead Encephalopathy.—Although lead is absorbed over long periods by workmen in mining towns without signs of plumbism, yet cases of intoxication appear from time to time in children, often with relatively short periods of exposure. The age incidence is dependent on the more frequent occurrence in childhood of acute febrile conditions with grades of acidosis severe enough to remobilize large amounts of lead from the bones. Byrom Bramwell, in his clinical studies, wrote:

> So closely do the two conditions (tumour and lead encephalopathy) in some cases resemble one another, that I never commit myself to a positive diagnosis of intracranial tumour without having previously excluded lead poisoning.

He might well have had in mind particular features of lead intoxication—the staggering gait, hypotonia and ataxia—which resemble the symptoms of cerebellar disorder, quite apart from the general signs of intracranial hypertension, including gross papilloedema and convulsions. There is no positive criterion of lead encephalopathy in children. For instance, no history may be available of access or exposure to lead in the form of accumulators, lead nipple shields, arsenate of lead fruit sprays, powdery lead paint on gates or verandas, or water from lead cisterns or pipes, and there may be no history of pica. In children with clean mouths the Burtonian lead lines will be absent. Wrist-drop and foot-drop are seldom seen in the young. There may be no hæmatological evidence of stippling or of cachexia. Lead may be found in the urine in amounts up to 0·1 milligramme per litre in apparently normal adults and children; this indicates only excessive absorption. Nevertheless, in most cases of intoxication figures of from 0·2 to 0·3 milligramme per litre are obtained on analysis. In the same manner X-ray pictures of the knee and ankle joints afford evidence of chronic absorption, dense "lead" lines appearing in the diaphyses about two centimetres distant from the epiphyseal lines of the radius, tibia and femur. However, these lines may be slight or absent, and in no wise indicate the presence of lead in a toxic form in the blood stream or brain. Thus diagnosis is based on the circumstantial evidence of excessive absorption, the presence of intracranial hypertension and the occurrence of major convulsions (the incidence of which is higher in encephalopathy than in cerebellar tumour). If prompt recovery does not follow adequate dehydration and the exhibition of calcium and phosphorized cod liver oil, suboccipital decompression is indicated, and examination should be made for evidence of basal arachnoiditis.

Third Ventricle Tumours.—Tumours in the region of the third ventricle may be either intraventricular or extraventricular in position. The latter include the suprasellar meningiomata, the optic chiasm gliomata, the anterior cerebral aneurysms, and the cranio-pharyngiomata, and frequently disclose their presence by field defects, calcification or erosion. As regards the intraventricular tumours, in Dandy's series approximately half the cases were undiagnosed before operation, and about one-quarter of the patients presented the cerebellar signs of dysequilibrium, staggering gait and unsteadiness in the Romberg position. In the majority of cases, then, the localizing signs are scanty and perplexing, and all that can be said is that the patient has increased intracranial tension. Symptoms are not always classically intermittent and dependent on the posture of the head, and it is of interest that Dandy himself finds it difficult to explain how obstruction to the ventricular system by actual shift of a ball-valve tumour could cause the precipitate onset of headache, blindness and dizziness. In ventriculography interest will be focused, as in all these cases, on the third ventricle, and particularly on lateral X-ray photographs taken with the patient's head in the hyperextended position according to the manœuvres of Lysholm, Twining *et alii*. McConnell and Childe point out that if the third ventricle is not filled, one is likely to mistrust one's technique, and thus fail to recognize the fact that posterior fossa lesions invariably cause such a pronounced dilatation of the whole supratentorial ventricular system that the third ventricle must fill unless occluded by tumour growth (this, of course, implies suitable and adequate manipulation and posturing). Radiographic findings include nonfilling, dilatation, displacements, partial fillings and deformities, and symmetrical dilatation of lateral ventricles depending on the patency of the foramen of Munro or on perforation of the *septum pellucidum*. Lateral ventricle tumours present the same type of problem.

Pineal Tumour.—In pineal tumour cases classical pneumographic signs are symmetrical internal hydrocephalus and a convex filling defect in the posterior half of the third ventricle with obliteration of the suprapineal recess. Of course, plain X-ray films may demonstrate pineal calcification, diagnostic of tumour in children, as it is not until after puberty that the involutional *corpora amylacea* make their appearance; they cast a shadow in the X-ray film in about 65% of adult people. Other fairly constant findings are the loss of pupillary light reflexes, inequality of pupils, difficulty in turning the eyes upwards and ptosis. Occasionally the *pubertas præcox* syndrome is present, and occasionally infratentorial extensions of the growth into the cerebellum produce false localizing signs.

Miscellaneous Group.—It has been found impossible to describe in detail numerous other conditions giving rise to the intermittent "H.V.P." syndrome. The most important of these may be mentioned—subdural hæmatoma, hypertensive vascular encephalopathy, stenosis of the aqueduct of Sylvius, and tuberculous meningitis.

Extracerebellar Lesions in which Cerebellar Signs and Symptoms Predominate.—Of the extracerebellar lesions causing a confusing preponderance of cerebellar signs, the infratentorial tumours achieve this result by direct pressure, whilst a different mechanism, probably interference with cortico-cerebellar pathways, obtains in supratentorial disorders.

Infratentorial Tumours.—Apart from the rare intramedullary tumours with their characteristic pharyngolaryngeal and facial palsies, and the occasional epidermoids and meningiomata clinically indistinguishable from intracerebellar tumours unless calcified, attention is directed to those tumours arising from Stibbe's "subtentorial angle". It is from here that acoustic tumours slowly and relentlessly extend, to impinge on the fifth and seventh bulbar nerves, to compress the cerebellum and to block

the incisura. Confirmatory tests add little to the classical anamnesis of progressive unilateral deafness, tinnitus, facial paralysis and trigeminal paræsthesia and anæsthesia.[1] Ataxias and signs of increased intracranial pressure, by the nature of the growth, must appear late in the history.

Supratentorial Tumours.—Of the supratentorial group, the parieto-temporal tumours are less prone to cause unilateral cerebellar disturbances. Kolodny recently described pseudo-cerebellar signs in only two out of a total of 38 cases of temporal lobe tumour. The association of homonymous hemianopia and incoordination occurring on the same side of the body in young people is very suggestive of parietal hydatid cyst, and cases such as these support the argument that "temporal ataxia" is brought about by interference with the temporo-ponto-cerebellar pathways.

In frontal lobe tumours, however, the so-called "frontal ataxia" of Bruns appears commonly. It appears unrelated to varying pathological conditions occurring in the presence of all types of tumour, in laceration and in brain abscess. The incidence, however, is high in surface meningiomata pressing directly on the premotor cortex; hence this phenomenon is attributed to interference with cells which form a link in the great integrating chain between cortex and cerebellum, and thus possibly to interference with the cells of origin of the fronto-ponto-cerebellar pathway (with this reservation, that dysequilibrium may be attributed to distant pressure effects on the nodulo-floccular lobes). There are certain continental neurologists who state that it is possible to differentiate at the bedside between "frontal" ataxia, "temporal" ataxia and cerebellar ataxia. Alfred Gordon, of Philadelphia, supports this view, and maintains that in frontal lobe involvement cerebellar signs are distributed irregularly to both sides of the body (a condition also seen in bilaterally expanding subvermis tumours), that the hypotonic element is usually lacking, and that progression on all fours is not rendered impossible by the ataxia. However, Frazier is among the many who consider that the associated signs and symptoms of frontal lobe tumour—the *Witzelsucht*, hebetude, forced grasping, incontinence, apraxia, seizures and hemipareses—are of greater importance in localization than the quality, degree or laterality of the ataxia. There are times, nevertheless, when the galaxy of frontal lobe signs is so vague, and the Bruns ataxic syndrome is so prominent, that the neurological surgeon might confidently perform a sub-occipital exploration were it not for a routine ventriculographic examination.

Conclusion.

The rational procedure, then, in the investigation of a child or youth who has complained of periodic headache and vomiting recently complicated by slight visual disturbances and unsteady gait, is as follows. In the first place, parents should be asked for additional information concerning head injuries, access to lead, holidays in sheep country (as immunological tests for ecchinococcosis are of little value in primary hydatid cyst of the brain) and recent sore throats, colds, toothache or discharging ears.

Secondly, examination should seek to confirm the existing evidence of intracranial hypertension. The eye-grounds should be examined, lumbar puncture should be performed, the pressure of the fluid should be carefully assessed and the fluid should be examined for protein and cell content. Stereoscopic X-ray films, preferably one pair of lateral and one pair of antero-posterior films, should be taken. As mentioned previously, these three procedures could be carried out at one sitting on uncooperative children by means of basal "Avertin" anæsthesia. If lead is suspected as an ætiological factor, the wrist and knee joints should be examined radiologically, the urine should be analysed quantitatively, and a blood count performed for the detection of stippling and anæmia. If the onset of the illness was associated with or aggravated by recurrent naso-pharyngeal infection or toothache, X-ray photographs of teeth or sinuses should be taken.

Finally, however faithfully and competently the interrogations, examinations and tests are carried out, it must be realized that many cases must remain in doubt until ventriculography has been performed. The interpretation of these ventriculograms may at times be exceedingly difficult and it is considered wise in all cases to be prepared to follow on immediately with major cerebral surgical procedures.

[1] The curious omission of involvement of the trigeminal nerve, particularly the absence of ophthalmic dermatome and corneal anæsthesia, observed by Cushing, was reported recently by Baker, Adson and Kernohan from the Mayo Clinic.

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THE SURGERY OF INTRACEREBELLAR DISEASE.[1]

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THE pathology of those intracerebellar lesions in which relief or cure may be obtained by surgical operation is restricted. Space-occupying lesions in the cerebellum fall into the two main groups of tumours and abscesses. Other

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neuro-Surgery of the New South Wales Branch of the British Medical Association on September 19, 1940.

intracerebellar conditions for which operation is indicated are rarely if ever encountered. Intracerebellar tumours are by no means uncommon, particularly in children, in whom the majority of intracranial tumours occur in the cerebellum. Cerebellar abscesses on the other hand are very rare, and as these are nearly all secondary to pyogenic infection of the mastoid air cells, their frequency of incidence is related to the effectiveness of drainage of the primary focus.

The surgical technique for the management of intracerebellar tumours and abscesses is now reasonably well standardized and presents no unusual problems once the pathology of the condition is recognized and its localization to the cerebellum is determined. In focal disease of the cerebellum, however, the localization of the lesion is often difficult. In a number of cases, fortunately the majority, the localization of the lesion is clearly written in the patient's symptoms and signs. The early appearance of headache, vomiting and papillœdema in association with hypotonia, ataxia, Rombergism and nystagmus, leaves little doubt as to its situation. This definitive group of signs is, however, by no means of universal occurrence, and in many cases the greatest difficulty may be encountered in the differentiation of a lesion situated in the cerebellum from others related to the posterior end of the third ventricle, the pineal gland and the frontal lobes of the cerebrum.

In a former contribution in this symposium, S. M. Morson has indicated the confusion which may arise between focal lesions in the posterior fossa and in other situations above the tentorium. Not only, as he remarked, may a cerebellar lesion be confused with a focal lesion elsewhere in the brain, but general conditions, such, for instance, as toxic hydrocephalus or lead intoxication, may not only produce gross increase of intracranial pressure with the typical manifestations of this disturbance, but may in addition modify cerebellar function and closely simulate the clinical picture resulting from focal intracerebellar disease. It is well recognized that patients exhibiting increased intracranial pressure and mild cerebellar signs require the most exacting investigation before it may be stated with reasonable certainty that they have a focal lesion in the cerebellum, and even when this investigation has been completed it is not infrequently necessary to tap both lateral ventricles and thus, by determining the existence of a bilateral symmetrical internal hydrocephalus, to provide evidence that the lesion is situated below the tentorium. Even this objective method is not completely adequate, as a similar hydrocephalic state is produced by a block in the posterior portion of the third ventricle or the aqueduct of Sylvius. However, disturbances in the last-mentioned situations may be excluded by methods which are not germane to the present consideration.

Summarily it may be stated that in a number of cases, particularly in children, in whom the cerebellar disturbance is more likely to occur in the vermis, the definitive localizing signs provide adequate justification for an immediate surgical attack on the posterior fossa. However, other cases are frequently encountered in which it is not possible to be certain whether the lesion is a focal one below or above the tentorium or whether the disturbances are the result of some general intracranial intoxication. In these cases the present writer regards it as a necessary routine preliminary to suboccipital operation to tap the posterior horns of the lateral ventricles and, if necessary, to make a ventriculogram and fill the whole ventricular system above the aqueduct of Sylvius with air before proceeding to a major operation. If this procedure is always adopted in cases of doubt, the unfortunate experience may be avoided of not finding a lesion in the posterior fossa at operation and subsequently discovering a tumour in one frontal lobe at autopsy.

The grave consequences of opening into the posterior fossa in the presence of a supratentorial tumour cannot be emphasized too strongly. The writer has had at least two narrow escapes. Some years ago a suboccipital craniotomy was performed after a diagnosis of left cerebellar tumour had been made in the case of a woman (E.N.), aged thirty years. After the dura had been opened, the cerebellum was found to be normal. The patient stopped breathing, and a needle was at once inserted into the lateral ventricle through an occipital burr hole. The patient's condition improved, and the suboccipital wound was closed. The error in localization was realized, and through a right frontal flap a cystic astrocytoma of the right frontal lobe was aspirated and removed. The great danger of respiratory failure occurs when a frontal tumour is mistaken for a contralateral cerebellar lesion and the suboccipital approach is used first. Fortunately, the opposite mistake is more common.

In another case, a girl (B.H.), aged fourteen years, had been prepared for a right cerebellar operation; but the preliminary insertion of a needle into the right lateral ventricle disclosed a hydatid cyst in the right occipital lobe, which, after being evacuated, was removed at a later operation.

Cerebellar Tumour.

In a recent article[1] the present writer referred to a series of cases of glioma of the brain. In the discussion of the cerebellar gliomata it was stated that over 90% of these are constituted by medulloblastomata and astrocytomata. Rarely in the cerebellum are encountered the astroblastomata or ependymomata, and more rarely still the hæmangiomata, which may be represented by a solid vascular tumour or by a small cyst with a microscopic mural nodule. The details of age incidence, situation, malignancy, radio-sensitivity and metastasis through the cerebro-spinal fluid were described; and it is proposed here to discuss briefly the methods of surgical attack and the type of result which may be expected.

In nearly every case of cerebellar glioma the patient has a high intracranial pressure with gross papillœdema and impending failure of vision, and may exhibit prodromal signs of herniation of the cerebellar tonsils in the *foramen magnum* or the uncus through the *incisura tentorii*. Dangerous impairment of visual acuity and impending medullary compression will not be avoided unless early examination of the *fundus oculi* is made in all cases when headache or vomiting is first complained of. Exigencies of time and the greater frequency of gastro-intestinal and other common causal conditions continually prevent such early detailed examination, and it is likely that for many years the neurological surgeon will find at the first examination of patients referred to him with cerebellar symptoms, high papillœdema and gross intracranial pressure. If respiratory embarrassment has already been produced by medullary compression, it is a wise procedure simply to make a burr hole in the occipital region and place a Frazier needle in the lateral ventricle, allowing continuous drainage of cerebro-spinal fluid for twenty-four to forty-eight hours; this will relieve the high pressure and consequently the respiratory disturbance before the posterior fossa is opened. If, on the other hand, the first approach is to the cerebellum, the simple uncapping of the posterior fossa by removal of its bony walls may determine a fatality before the lesion is seen. Tapping of the lateral ventricle will not unlock an existing herniation through the *foramen magnum*, but it will prevent it; the writer has treated patients who, first seen when they were passing into coma, have become conscious and rational within a few minutes after a Frazier needle has been placed in the lateral ventricle. It is important, however, in these cases of posterior fossa lesions, to operate shortly after relief of pressure by this means, since the growth may cause hind brain displacement and the patient may die suddenly in spite of a normal cerebro-spinal fluid pressure.

As an example, a patient, H.S., was referred last year to the neurosurgical department at the Royal Prince Alfred Hospital with a diagnosis of cerebellar tumour. The patient was in coma on his admission to hospital, and was immediately taken to the operating theatre, where a Frazier needle was inserted through an occipital burr hole into the lateral ventricle. The patient became conscious within one hour of this operation, and during the next twenty-four

hours was quite rational and cooperative, and taking food and fluid by mouth. Operation had been planned for the next day, but owing to the occurrence of a streptococcal throat in one of the theatre staff the operation was postponed for twenty-four hours. The Frazier needle was left *in situ*; but the patient died suddenly that night, and at autopsy a small astrocytomatous cyst with a mural nodule was found in the left cerebellar hemisphere.

In a typical case, after the lateral ventricle has been tapped, the posterior fossa may be aproached by a mid-line nuchal incision, which permits removal of the posterior border of the *foramen magnum* and the arch of the atlas with relatively wide exposure of the vermis and cerebellar hemispheres. Vermis tumours may be approached through this incision, and if on the other hand the tumour is found to be in one hemisphere, a suboccipital muscular flap may be reflected on that side and access may be obtained more laterally, even into the cerebello-pontine angle. The value of the mid-line approach lies in the rapidity with which the cerebellum may be exposed, in the simplicity of closure and in the bloodless field which results from incision through the *ligamentum nuchæ* by endothermy.

When the cerebellum is exposed the tumour only rarely presents on the surface, and in nearly every case it is necessary to pass a brain needle first into the vermis and then into the hemispheres before its situation may be accurately determined. Subsequent surgical procedure is conditioned by the results of the insertion of the needle. If the needle strikes a solid tumour in the vermis, small fragments of the tumour may be aspirated into the needle and examined histologically. When the tumour is solid and situated in the mid-line, particularly in children about the end of the first decade, it is usually a medulloblastoma. These tumours are malignant, cannot be totally removed by operation, inevitably recur within twelve to eighteen months of operation and are very prone to form drop metastases down the spinal cord if they are sectioned. They are, moreover, perhaps the only gliomata which are really sensitive to deep X-radiation. The present writer believes, therefore, when such a tumour is identified, that it is wise to terminate the operation after adequate decompression, and subsequently to treat the patient with intense deep X-ray therapy. This method not only produces an equally satisfactory immediate result, but in some cases the deep X-radiation alters the histological character of the neoplasm in a manner which enables it to be separated from the surrounding cerebellum at a subsequent operation and offers a better opportunity for its complete extirpation when symptoms recur.

A patient (H.R.) was referred to the writer from Brisbane in 1939, and a solid medulloblastoma was found occupying the right side of the vermis and most of the right cerebellar hemisphere. This patient was treated with intensive deep X-radiation. At a second operation six months later the tumour was again exposed and was then found to be relatively avascular; it was removed *en bloc* by cotton-wool dissection from the surrounding cerebellum.

If the insertion of the needle, on the other hand, discloses a tumour in one cerebellar hemisphere, particularly if it it cystic, it is almost certainly an astrocytoma, and as these growths are relatively benign and since many of them may be completely removed, a radical surgical procedure is indicated; this implies in many cases the removal of all or nearly all of one cerebellar hemisphere. Sometimes the surgeon is fortunate enough to encounter an astrocytomatous cyst containing a small mural nodule, which may be completely removed if the cyst is opened without the necessity of excision of the surrounding cerebellum. Apart from the technical difficulties, however, removal of more or less the whole of one cerebellar hemisphere is of no great consequence, and does not subsequently impair even the finer coordination of the ipsilateral limbs.

In a recent case, during the removal of a cerebellar cyst the surrounding cerebellar tissue was removed so deeply that an opening was made into the fourth ventricle. The patient (G.H.) recovered completely and recently returned to his duties as a bank officer.

Survival periods following operations on medulloblastomata are universally short, and it is unusual to find a patient with a cerebellar medulloblastoma alive five years after operation in spite of intensive deep X-radiation. The outlook with astrocytomata, however, is much better and survival periods of fifteen to twenty and more years have been described.

To take a long range view of cerebellar gliomata, it may be said that they offer either the best or the worst ultimate operative results. The operative mortality rate in these cases is relatively low and the immediate result is invariably good. Provided that the patient is referred for treatment before medullary compression becomes imminent and that the surgeon is careful to relieve excessive intracranial pressure before opening the posterior fossa, the operative mortality rate should not exceed 10%. The survival periods may range from two to twenty years or more, depending on the histopathology of the tumour; and as this cannot be determined with certainty before operation, the tumour should be exposed in every case and the subsequent procedure determined by the result of macroscopic or microscopic examination.

Cerebellar Abscess.

Cerebellar abscess is at least three time less common than supratentorial abscess, and in nearly every case it is preceded by septic infection of the ipsilateral mastoid air cells. Diagnosis and localization present no unusual difficulty, particularly since examination of the cerebro-spinal fluid in these cases will invariably reveal a protein content greater than 30 milligrammes *per centum*. It is quite safe to remove one cubic centimetre of cerebro-spinal fluid for this purpose immediately before operation. As with tumours of the cerebellum, the lateral ventricle should be tapped immediately before operation and the high intracranial pressure reduced by transventricular drainage. The time to operate is of fundamental importance and has been discussed already both by Atkinson and by McEwen. As a general working principle, the longer the patient is able to retain the abscess without the appearance of signs of compression of the hind brain, the more certain will his ultimate recovery be; it is foolish to attempt to drain an abscess which has no definite glial wall, since meningitis will be a common result. The danger of this complication has been reduced in streptococcal infections by the introduction of the sulphonamide drugs. However, it will always remain a danger to be avoided. There are three schools of practice in the technique for the surgical treatment of abscess of the brain—namely, aspiration, continuous drainage and marsupialization. Dandy prefers simply to aspirate the abscess through a wide-bore needle after removal of the overlying bone. King, after removing the overlying bone, uncaps the abscess, evacuates the contents and lightly packs the cavity, leaving the wound open. Others pass a tube or a catheter through the overlying brain and suture this tube to the edges of the wound, allowing the abscess to drain into a doughnut dressing for several days, then by daily dressing gradually remove the tube a few millimetres each day. Special difficulties are present in cerebellar abscesses owing to the proximity of the lateral and sigmoid sinuses and the possibility of sinus thrombosis. Marsupialization is difficult in that situation, owing to the mass of suboccipital muscle overlying the bone defect. It is not possible, therefore, to describe any method of election, and each case must be treated on its merits according to the persistence of mastoid infection, the presence of sinus thrombosis, the degree of intracranial pressure and the amount of general intoxication of the patient. A conservative attitude in these cases is always a wise one; even if the patient is very ill, a successful result may more frequently be obtained by continuous transventricular drainage and sulphapyridine therapy in streptococcal infections than by an immediate attempt to aspirate or to drain the abscess. In any case, the condition is always hazardous and the surgeon may count himself fortunate if he finds among his records one or two patients suffering from cerebellar abscess who have been successfully treated by operation.

Reference.

(1) G. Phillips: "The Management of Cerebral Gliomata", THE MEDICAL JOURNAL OF AUSTRALIA, October 5, 1940, page 301.

EXTRACEREBELLAR TUMOURS.[1]

By I. DOUGLAS MILLER,
Sydney.

THE class of lesion with which we are more directly concerned is the extracerebellar space-occupying lesion in the posterior fossa. It is often a difficult matter to hazard an opinion as to whether a tumour is inside or outside the cerebellum, and though it may at first sight appear to be a rather academic point, one must bear in mind that the neurological surgeon does well to visualize every detail of pathological anatomy in the planning of his operative attack. Considerations of age have an important bearing. Infants and children are subject to the intracerebellar neoplasms, and the only extracerebellar tumour I have seen is a dermoid cyst, which is such a rarity that I am unlikely to see another. This lesion was looked upon as possibly an astrocytoma, and there was nothing in the history or signs to indicate otherwise, though reference to the X-ray film reveals an appearance which I have not seen associated with intracerebellar lesions.

A boy came to me a few weeks ago with a history and a clinical picture suggestive of a cerebellar astrocytoma; but in the X-ray film there was a little definite calcification, which emboldened me to diagnose a dermoid cyst. Operation, however, disclosed a large solid tumour of the cerebellum, extending down onto the fourth ventricle. This tumour—of the astrocytoma class—showed numerous small foci of calcification.

In adult life the extracerebellar space-occupying lesions are tumours of the nerves, the most important being the eighth nerve; less frequent are tumours of the fifth nerve, cystic arachnoiditis, meningiomata, and metastatic neoplasms.

It is a fairly true rule that any posterior fossa tumour in a person aged over fifty years will be an acoustic neurinoma.

These tumours are usually readily enough differentiated from intracerebellar tumours by reason of the involvement of peripheral nerves, though of course massive intracerebellar tumours may affect nerves, notably the third and the fifth.

As acoustic neurinomata are the most common of all these lesions, I shall use them as my text. Every neurologist is familiar with the classical picture of these tumours; there is a history usually extending into years, ushered in by tinnitus and deafness, which may be so gradual as hardly to be noticed and often completely forgotten in the subsequent relation of the history. The next thing noted is an instability in walking and a clumsiness of movement. The unfortunate sufferer is usually regarded as a quiet tippler; a later development is headache and vomiting. In addition to these classical three steps, there are neighbourhood signs in the peripheral nerves which are variable and inconsistent. The nerve most commonly secondarily affected is the seventh, which may be completely paralysed or may merely show some occasional weakness. The fifth nerve is frequently affected, as shown by anything from neuralgic pain and anæsthesia to a mere loss of the corneal reflex. The sixth nerve may be manifestly damaged. Much less commonly the nerves issuing below the internal meatus are affected, and there is difficulty in swallowing or hypoglossal paralysis. In addition to these signs pronounced spastic paresis of the limbs on one or other side is often seen.

The pathological march of events bringing about this picture is readily understood. First, the eighth nerve is destroyed, then as the tumour grows it pushes into the cerebellar hemisphere. Continued progress leads to dislocation of the brain stem and the onset of internal hydrocephalus, whereas extension outwards into the angle or along the acoustic meatus leads to the peripheral nerve palsies. The motor and sensory interference often experienced in the limbs is due to pressure of the brain stem against the incisura. This is most likely to have the effect of compressing the heterolateral tracts and so of giving a homolateral set of signs in the limbs, though the reverse may happen also. The whole picture as sketched is rarely present, and particularly in early cases we may see one little set of features dominating the picture and leading possibly to great confusion. Every clinician has been sorely tried in deciding whether some of these lesions are in fact space-occupying angle tumours or disseminated sclerosis. I am emboldened by having many good companions in my error to confess that I have made the mistake both ways. I have come now to the working rule of not seriously considering a diagnosis of posterior fossa tumour until papillœdema is present. It is not a fact that a ventriculogram will clear up this doubt, for often in degenerative diseases such as disseminated sclerosis there is considerable ventricular enlargement, which may merely lead to the assumption that this is due to a block. Moreover, let it be remembered by anyone who considers the problem simple that the papillœdema noted may in fact be a phase in the active optic neuritis of disseminated sclerosis. These patients often present a picture of diffuse brain damage, being semistuporose. It is in such cases that one is very apt to miss the point, as the surgeon may not get a history of deafness and may well not note it. Such lesions are often mistaken for subdural hæmatomata, which may give the same ill-defined picture. Coma of this sort may come quickly into the picture owing to sudden softening of the growth or cystic collections around it.

This is no place to discuss the further difficulties of these tumours which seem to fight against cure on every aspect. It is a strange paradox that the most benign of tumours can give the greatest trouble in removal. Surgeons have tried all ways of dealing with this tumour, and the fact that every modification of technique and every new suggestion is eagerly tried the world over bears testimony to the difficulty and danger of their removal. I mention this, as people not conversant with the facts are apt to think that there could be no tastier morsel for a neurological surgeon than a little acoustic neurinoma, whereas in fact this tasty morsel constitutes usually the major battle of the surgical craft.

These tumours are acoustic neurinomata or schwannomata, and as such are among the most benign of neoplasms. They should be ideal for radical removal; but such attempts are exceedingly dangerous and inevitably mean the sacrifice of the seventh nerve, for they originate well within the acoustic meatus, and even though the seventh nerve may not clinically be severely implicated, surgical removal of the tumour will mean complete division. In order to remove the tumour adequately the outer third of the cerebellum should be removed. This means a considerable disability from which it may take years to recover. As these tumours are so apt to bury themselves in the brain stem or push up through the incisura of the tent, the technical difficulties and dangers of removal are great, for hæmorrhage from the tumour bed will constitute a severe hazard, pressure in this region being out of the question, and the endotherm—that great standby of the surgeon—highly undesirable in this locality. The serious dangers of such an operation and the inevitable sequelæ of complete facial palsy and possibly a worse ataxia than the patient had before, may well serve as argument for less than complete removal.

Cushing, after many attempts at complete removal, came back to intracapsular removal. This less than complete operation achieves decompression, and the tumour is removed from within its capsule. This protects the seventh nerve and does not necessitate cerebellar damage. However, post-mortem examinations made after what has satisfied a surgeon as being a good intracapsular removal usually show how incomplete it has been. Recurrence is inevitable, though it may not take place for years, and subsequent operation will be more difficult than before.

[1] Read at a meeting of the Section of Neurology, Psychiatry and Neuro-Surgery of the New South Wales Branch of the British Medical Association on September 19, 1940.

The acoustic neurinomata are usually not relieved by a simple posterior fossa decompression, for the anatomy of their compressive effects differs from other posterior cerebellar fossa tumours in not causing a herniation of cerebellar tonsils and so a low block to the cerebro-spinal fluid; but it causes its pressure either by direct aqueduct kink or by being forced through the incisura. This is a matter of importance if less than total removal is decided upon, as a decompressive effect will not be achieved until this upper extension beneath the tent is dislocated or removed.

It is in this dilemma that the most benign of tumours places the surgeon, and I feel that it necessitates his putting the cards on the table with the patient or his people, and to a large extent acting in accordance with their wishes.

I shall not now discourse on the other posterior fossa nerve tumours, which are rare, or on meningiomata or metastatic tumours, all of which may present the same type of difficult neurological and surgical problems from the aspects of diagnosis and treatment.

Reviews.

"MYTHOLOGY OF THE SOUL."

DR. H. G. BAYNES, the author of "Mythology of the Soul",[1] commences his large volume with a quotation from Tao Te Ching which is well worth repetition:

> The Sage's way of curing disease
> Also consists in making people recognize their diseases as diseases and thus ceasing to be diseased.

This particular sage (Dr. H. G. Baynes) uses dreams and drawings to make his two schizophrenic patients reveal themselves to themselves and so aid self-cure. The case material in the text is woven into an argument for the collective unconscious.

The author is loyal to Professor Jung, whom he regards as the yellow ancestor of analytical psychology. His followers must either admit their indebtedness or else refuse to admit his existence.

The chief causes for schizophrenia are regarded as either the lowering or weakening of the conscious control in face of stress or the excess activity of archaic contents in the unconscious. Dr. Baynes contends that a consideration of this balanced action enables us to understand our present world affairs. In the mind of the average citizen is a renegade voice in his psychic make-up. This gives an instinctive appeal to lawlessness, which is condoned as a virtue in the crown of nationalism.

The author writes well, but is he not cursed with the facility of the too-ready writer? Discussing sex in schizophrenia, he states: "It is the inferior type of individual who pursues the fata morgana of sexual perversity, goes whoring after magic or yogi-cults, or traffics in occultism with purveyors of cheap mystery." Yet interspersed through the 900 pages of text are serious references to such cults in discussions on the evidence of the archaic unconscious. To run with the hare and hunt with the hounds may be a common practice, but it is indefensible in a psychiatrist. It may be argued that an extension of this argument provides the strongest evidence against the truth of the Baynes theme.

The first case notes concern a physician who after an unhappy domestic and married life developed a psychosis. The analysis commences with a dream which is admitted to have coincided with his reading of Freud's "Interpretation of Dreams". The author naïvely remarks: "An experience which had given his mind a very definite orientation and which opened his eyes to the possible value of dreams."

Is it remarkable that a zealous student steeped in sex-tinted psychology should draw a series of coloured tableaux in which crude symbolisms of the penis, uterus and semen type predominate? In view of man's innate suggestibility, it would seem almost axiomatic that any research into the archaic material of the unconscious should be conducted in patients uncontaminated by previous psychological reading.

Perhaps the most striking drawing of the archaic is that of an "eight-trunked" elephant which shows remarkable resemblance to the white elephant of the Muladhara Chakra of Indian lore. This animal had seven trunks, and although the patient is stated never to have seen the mythological prototype, it is questionable whether such a denial is to be taken seriously, and if not, whether the possibility of coincidence can be excluded.

In similar terms may be criticized the alleged resemblances to drawings of T'ai Ch'i or Great Monad and the characteristic lamaistic ornamentation of Tibet.

The final series of drawings and paintings reveal considerable artistic ability. The author's description of one of these, number 23, is:

> The scene is under the sea. The blue depths are illumined by a candle which burns with a steady flame near the centre of the field. Attracted to the flame a shoal of small fish are seen swimming from the right into the orbit of light. These tiny fish are done with great delicacy. Some nearest the light bear a bright orange spot on their sides, as though reflecting the flame of the candle.
>
> Circling the candle with leisurely power are two sharks, one of them lying north-west of the candle, the other south-east. Their position, equidistant from the candle in the centre, suggests a relation of polarity, and their curving motion suggests that they are circling slowly around the candle. The patient told me he had not intended them for sharks, but he agreed that sharks they undoubtedly were and suggested that their circling around the flame was a kind of dance.

The "solution" concerns the patient's appreciation of a current problem. He has solved a domestic difficulty. "A light has been lit in the depths of the unconscious and because it burns in the sea it cannot be quenched." But does it follow that as the candle is the archaic means of light production, the drawing is evidence of the truth of the Jungian unconscious? Was it chosen for its phallic significance or for its æsthetic appeal, or was it just a coincidence? There are many possibilities. Whilst the author makes a clever and well-documented case for the collective unconscious, the urge for logic is not satisfied. It is an argument from the particular to the general and, as already mentioned, the evidence is tainted at its source.

Archæology has been impressed on the matrix of the mind as an artefact; its rediscovery may reveal no more than old thoughts in a new setting.

Perhaps in another volume the author may describe the drawings of less mature minds and will be able to control the ingress of outside ideas. Until he can do so, his theory is limited in its scope and must fall under the ban of scepticism.

The opening chapters give an excellent résumé of psychological fact and theory. It is apparent that the author is well conversant with his subject. His inability to prove the truth of his theory must be ascribed to his material and certainly not to his lack of courage. An excellently printed and illustrated work of this scope and dimension printed in war-time is surely an event if not an epic.

[1] "Mythology of the Soul. A Research into the Unconscious from Schizophrenic Dreams and Drawings", by H. G. Baynes, M.B., B.C.; 1940. London: Baillière, Tindall and Cox. Medium 8vo, pp. 952, with 53 illustrations. Price: 32s. 6d. net.

DEDUCTIVE ANATOMY.

A REPRINT within twelve months and a new edition within three years, fully justify the good opinion entertained about the first edition of Boileau Grant's "Method of Anatomy".[1] The new edition contains nearly 150 additional pages, of which about half are occupied by an introductory section on systematic anatomy. The idea of this preliminary general survey is good, but it scarcely warrants inclusion of such purely physiological considerations as supplemental air and complemental air; and physiologists, no doubt, will question the figure of 2,500 cubic centimetres for complemental air—a litre higher than the amount usually given. The remainder of the book follows the lines of the first edition, but is expanded and more copiously illustrated. Some of the objections to the first edition have been removed—the surgeon is not expected to be so optimistic about his prospects of curing a femoral hernia; on the other hand, sterno-cleido-mastoid survives, but perhaps this is a subconscious protest against nomenclature revisions. This book will continue to grow in popularity.

[1] "A Method of Anatomy, Descriptive and Deductive", by J. C. B. Grant, M.C., M.B., Ch.B., F.R.C.S.; Second Edition; 1940. London: Baillière, Tindall and Cox. Crown 4to, pp. 816, with 651 illustrations. Price: 33s. net.

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MEDICINE AND THE FUTURE: A VOICE FROM NEW ZEALAND.

If the present war has done nothing else, it has made many people realize that change is bound to come in many aspects of communal life, and all who have studied the subject know that the practice of medicine will be amongst those that will see great changes. This subject has been mentioned more than once in these pages, and amongst the authors who have discussed it are A. E. Brown, of Colac, Victoria, Stanley Boyd, of Gnowangerup, Western Australia, and W. P. Holman, of Launceston, Tasmania. Our contention has always been that since changes are bound to come, the medical profession should recognize that they are inevitable and should take the lead in directing the changes along lines that will make every advance in medical science, every preventive agent of proved worth and every reliable therapeutic weapon available to every member of the community, that will leave medical investigators free to pursue research in whatever direction they feel urged to move, and that will be consonant with the dignity and weal of medical practitioners. The latest to enter discussions on this subject is Douglas Robb, of Auckland, New Zealand. Robb is known to the surgically minded practitioners of the Commonwealth by reason of his contributions to surgical literature; but now in his book, "Medicine and Health in New Zealand",[1] he appears before a wider public as a man of thought and insight who is not content to drift and to allow things to happen round him as they will, but wishes to bring to the difficulties of the future constructive contributions based on knowledge and experience. He writes in his preface: "That any changes made here should be wise ones, and in the best interests of the people and of the progress of medicine, is my only concern. I believe in medicine as an instrument of human welfare. . . . All sections of the people must understand and agree to any changes that are decided upon. If then my words seem addressed to my professional colleagues as much as to the people in general, it is because we have a special responsibility. A cynic once said 'We are not the doctors, we are the disease'. We can best prove his error by taking the lead in reconstruction."

The publication in New Zealand of a book such as Robb's is the more interesting on account of the *Social Security Act* which was passed by the New Zealand legislature in 1938. According to the scheme on which this act was drafted every person in the Dominion was to be entitled free to the services of a general practitioner and to hospital, sanatorium, mental hospital and maternity treatment, and later when practicable to the services of specialists, dentists, opticians and home nurses. The scheme covered old-age and invalid pensions as well as payments to widows and orphans. It was estimated that the cost would be something in the neighbourhood of £16,000,000 a year. Quite naturally, such far-reaching proposals aroused a great deal of opposition from the members of the medical profession, for it was realized that the proposals involved socialization of the medical profession in advance of socialization of the whole community. *The British Medical Journal* pointed out at the time that the people of New Zealand had not decided to adopt socialism for the whole community and that there was no reason why the medical profession should be chosen for such an experiment. Readers should be reminded, however, that the New Zealand Branch of the British Medical Association made it clear that it was willing to accept and cooperate in a system which offered a complete medical service to those members of the community who were not in a position to secure it for themselves—"an offer of very great value and importance, far removed from any selfish or obstructive motive". There is no need to attempt to set out in detail any of the discussions or negotiations of the last year or two. Suffice it to state that hospital benefits under the *Social Security Act* are in operation as well as maternity benefits that receive the approval and cooperation of the medical profession. The medical benefit has not yet been inaugurated. It is in this present state of affairs that Robb enters the arena with his plea for reconstructive effort.

It is not our intention to review Robb's book in the usual way, but rather to use his arguments to show once more that consideration must be given to the future. If in so doing we can persuade those interested in the subject to study his book, part of our object will have been achieved. One of the most valuable aspects of Robb's book lies in what may be called his survey of medical practice, particularly general medical practice, as it is carried out in New Zealand. In Great Britain many of those who are prepared to lay down the law on medical practice in any and every place do not realize what medical practice in a country such as Australia is like, with its large city centres on the one hand and on the other its country centres that present enormous variations in regard to population, climate and distance one from another. In the same way Australians cannot know a great deal about New Zealand conditions, though they will presume that in many respects they will resemble those of this Commonwealth. Since there is in fact much similarity, Robb's remarks may well claim our attention. True of other countries as well as of Australia and New Zealand is his statement that the people must realize their importance as both providers and consumers of health service. "They must not only

[1] "Medicine and Health in New Zealand: A Retrospect and a Prospect", by Douglas Robb; 1940. Auckland: Whitcombe and Tombs Limited. Demy 8vo, pp. 146. Price: 8s. 6d. net.

demand the best; they must provide the means to achieve the best." Though the freedom of personal initiative that is theoretically enjoyed at present is becoming "more and more frustrated" in health services "by the contradictions that have grown up within them", Robb thinks that "freedom can be restored and made real only by the acceptance of a greater degree of organization along functional lines". In this organization experts must be given authority and the needs of the people must remain paramount. In every department of medicine, the basic sciences and the strictly medical subjects, development is taking place in an extraordinary fashion. Old knowledge and attitudes no longer tenable must be abandoned and all things have to be considered in the light of new knowledge. But Robb insists that in all this world of flux one thing at least remains unchanged and this is the scientific method. The scientific basis of medicine can, he holds, be ignored only at the price of stagnation and failure. Not all groups in the practice of medicine and not all hospitals accept scientific standards. Robb points out that a community or a hospital usually has to reach a certain size or maturity before it feels that it can undertake more than routine work. He is not satisfied with the attitude of New Zealand graduates either in the practice of curative medicine or in that of preventive medicine, aptly described as embracing the whole study of man—"by the time the New Zealand graduate has become established in practice, in whatever branch of the profession, he is rarely haunted by scientific aspirations". Robb also thinks that concentration on curative work is responsible for many of the practitioner's troubles. The difficulties confronting the family physician are many and varied and mostly lie beyond his control. They arise, Robb believes, from faulty preparation for the work of medical practice and from the disproportionate rewards obtainable in its several branches. "This faulty emphasis is, of course, merely the medical aspect of a fairly general disorder of our contemporary life."

It would be an impertinence on our part to offer any criticism on Robb's specific references to New Zealand practitioners. That the scientific spirit does exist in the Dominion is evident from the contributions to medical and surgical literature by New Zealand practitioners. At the same time, the retention of a scientific outlook is made difficult for New Zealand doctors if hospitals generally are subjected to anything like the deplorable conditions of management described by Robb in regard to the Auckland Hospital. These considerations need not detain us; what should be noted is that much of what Robb writes about New Zealand practice is applicable to Australia. This fact becomes the more significant when Robb tells us that it is over the scene described by him that the shadow of State organization is hovering. We need not concern ourselves with any remarks that Robb makes about the British Medical Association in its handling of the proposals made in connexion with the *Social Security Act*. He thinks that the institution of a salaried service for general practitioners might meet all the difficulties discussed by him. In support of this he states that in such circumstances (*a*) competition for patients and paying work would vanish; (*b*) plans could be made to serve needs not at present being served at all, or being only poorly served, by the setting aside for them of individuals of appropriate temperament and interests; (*c*) each kind of work could be done by men and in places best suited for the purpose; (*d*) the individual worker would be free to direct all his energies to the work in hand, unhampered by the tedium of "keeping up appearances" and collecting his money. These are arguments which we have heard before; we have also heard the objections that a salaried service will lead to place seeking and struggle for advancement and an early loss of interest in work, and that red tape and paper work are inseparable from such a service. The possible safeguards that Robb sees to such objections includes, first of all, the fact that medicine is a good mistress and that there are immense satisfactions to be derived from work for its own sake. Further, standards for promotion can be adopted and nepotism and favouritism denied. What Robb really wants (and this is the main purpose of his study) is that the appropriate authorities should make a detailed survey of the whole field in as leisurely a manner as is necessary for thoroughness. He holds that suitable surveyors are to be found in New Zealand and amongst New Zealanders; one of his wisest contentions is that amongst the surveyors should be at least some whose interest lies in the future rather than in the past. Robb has set out the features that in his opinion should be considered whatever the changes may be that are finally undertaken. They are as follows:

1. Reconsideration of undergraduate training for men and women destined for general practice.
2. Provision for regular periods of post-graduate study.
3. Library and journal service.
4. Mutual relief amongst doctors regarding night work and week-ends.
5. The attachment of every general practitioner to some large institution or organization, according to the nature of his work, for his help, encouragement, and criticism. Examples are diagnostic clinics, hospitals both general and special, and Government departments such as the Education Department.
6. A salaried service, with the usual accompaniments, such as retiring allowance, regular leave *et cetera*, might be considered for general practitioners. If decided upon, this could be introduced either gradually, by making appointments to country districts at present ill-served and by filling other gaps, or more abruptly. Experience would show how slowly or how rapidly the transition was desired by the people and by the doctors.
7. Once the people have decided the form the service is to take, medical men should be placed in charge of its management. This sound principle was used in the medieval guilds, and is conducive to high standards of work and smooth operation.

From what has been written it is clear that Robb has tried to state his case with clarity and fairness; he is certainly in earnest, and though he appears to think that a fully salaried medical service will best serve the interests of the community, medicine and the medical profession, he does not specifically say so. We need not comment on his plea for a survey in New Zealand. What does concern us is his argument and his general outlook. No such legislation as has been framed in New Zealand has as yet been attempted in Australia; we do not know when the attempt may be made. If legislation dealing with the practice of medicine is introduced wise counsels will need to prevail among medical practitioners. Robb has shown Australians what considerations will have to be faced and his book should be read from this point of view as well as for its general interest. The Federal Council of the British Medical Association is considering a scheme of medical service for the nation. If the members of the

several Branches are wise they will approach this scheme with the idea of service uppermost in their minds. The will of the people must prevail in the long run and organized medicine, recognizing this fact, should take the lead in trying to influence public thought. In this regard the several Branch councils would do well to seek from among their Branch members persons who would be suitable candidates for election to future parliaments, so that medicine may have fuller representation in our legislative chambers than it has at present. That, however, is a large subject and its discussion must be left for another occasion.

Current Comment.

THE ANATOMICAL BASIS OF GASTRIC ANACIDITY.

THE absence of hydrochloric acid from the gastric juice is by no means uncommon, and in the past a number of causes have been put forward to account for the phenomenon. It is well known to occcr in certain organic diseases of the stomach, such as carcinoma. There is strong evidence too that psychic stimuli can produce achlorhydria. Pavlov demonstrated this in dogs. Heyer and others showed that when human beings were hypnotized and then subjected to emotional stimuli, a temporary gastric anacidity could be produced, and it has since been shown that this functional aberration is sometimes resistant to histamine. There seems little doubt, then, that a psychogenic achlorhydria, at least temporary in nature, does occur. A number of workers have claimed that the condition exists as an inherited constitutional abnormality in otherwise healthy people. They have cited the occurrence of symptomless achlorhydria in groups of healthy young adults, and particularly in the families of patients suffering from pernicious anæmia. The significance of the finding in this latter group was discussed from a different aspect in these pages on January 18, 1941. In a recent discussion of the anatomic basis of gastric achlorhydria, R. Schindler, P. B. Nutter, H. E. Groom and W. L. Palmer[1] question the existence of this inherited physiological anacidity. Having carried out gastroscopic examinations on a large group of achlorhydric patients, they claim that a stomach cannot be declared healthy unless examined in this way, and that the anacidity in allegedly healthy individuals may in fact be due to a relatively symptomless chronic gastritis. It must be agreed that their findings lend support to such a claim.

Gastroscopy was carried out in a group of 101 patients with spontaneous achlorhydria resistant to injections of histamine on one or more occasions, and in 19 patients in whom the disturbance of gastric secretion resulted from some artificial interference. Of all these patients only five were found in whom organic disease of the stomach could not be discovered through the gastroscope. Of these five, one was suffering from chronic bismuth poisoning resulting from antisyphilitic treatment, and might have been expected to have gastritis, though it was not macroscopically visible. In the other four the achlorhydria may have been psychogenic, though this could not be proved, nor was it known whether the condition was constant or temporary, for only one histamine examination was performed in each instance. In 55 patients a diffuse gastritis was found. In six it was hypertrophic in type, in 12 superficial, in seven superficial but tending to become atrophic, and in 30 frankly atrophic. The proportion of examples of atrophic gastritis in this group is considerably higher than is usually found by gastroscopy of patients examined without consideration of the existence of achlorhydria. It seems that this functional disturbance tends particularly to be associated with the atrophic form of chronic gastritis. In none of the patients with the hypertrophic form of the disease was the achlorhydria proved to be permanent, and in those in whom repeated histamine examinations were made the gastric acidity was found to vary quite widely, acid often being present in considerable amounts.

Sixteen patients with pernicious anæmia and anacidity were examined. In twelve chronic atrophic gastritis was found and in two a gastritis of a mixed atrophic and superficial type. In the remaining two patients no macroscopic lesion could be detected, but they had been under hæmopoietic treatment for some time and it is known that this may effect an improvement in the condition of the gastric mucosa. Examinations of particular interest were carried out on three achlorhydric but otherwise apparently healthy relatives of patients suffering from pernicious anæmia, and in all three a severe atrophic gastritis was found. The authors suggest that the achlorhydria in these patients is not a familial trait, but a result of the gastritis. We would suggest the possibility of the reverse relationship, that the achlorhydria was inherited and the gastritis the consequence. Among 23 patients with malignant tumour of the stomach and anacidity many examples of severe atrophic gastritis were found. These findings are in accordance with the concept that chronic gastritis and acid deficiency may precede the formation of a carcinoma, and that the anacidity so often found in cancer of the stomach is in fact due to the gastritis. In three of these patients, however, a cancer was present, but no macroscopic evidence of gastritis was found, and the suggestion is made that "toxins" from the cancer caused the suppression of the acid secretion.

Among the patients in whom the anacidity was thought to have been produced by artificial interference, eight had had extensive operations on the stomach; six had had partial resection and two gastro-enterostomy. All had severe diffuse gastritis. In two it was known that the gastric secretion was normal before operation, but after operation a severe purulent gastritis with anacidity developed. It was thought that the anacidity was due at least in part to the gastritis and not solely to the reflux of intestinal alkalis. The authors point out that it is known that in dogs a very severe gastritis regularly follows operations on the stomach. X-ray therapy was the artificial interference in eleven other anacid patients, and all of these were found to have a macroscopic gastritis, varying from a mild superficial and temporary lesion to a severe chronic atrophic one. Neither the gastritis nor the achlorhydria was permanent in all these patients.

Schindler and his colleagues have presented an impressive array of facts to support their thesis that anacidity is almost always due to an organic lesion of the stomach, most often an atrophic gastritis, of such degree that it can be demonstrated macroscopically by the gastroscope. They have performed a valuable service in emphasizing the value of the gastroscope in investigating abnormalities of gastric secretion. The picture, however, is as yet by no means complete. Their case is weakened by their failure to state clearly whether their patients complained of digestive symptoms that led to the discovery of the secretory deficiency. If they did, it is by no means surprising to find organic disease present, and the report is no proof that achlorhydria is usually a sequel to such disease. Nor does the report help us greatly to decide whether the organic disease causes the achlorhydria or *vice versa*, or whether they are coincident conditions arising from some common cause. One important thing, however, does emerge, that achlorhydria is often associated with organic disease of such a nature that it can be demonstrated only through the gastroscope. The relationship that these two features bear to one another can be decided only by more extensive surveys. We would suggest that gastroscopy should be added to histamine gastric analysis in the examination of groups of apparently healthy and symptomless people, such as medical students. In some of these it is highly probable that either achlorhydria or gastritis would be found, separately or in combination. If they are found in combination, additional evidence is added to that contained in this report; if separately, repeated examinations will reveal whether the two conditions can persist for long as separate entities and, if not, which is cause and which effect.

[1] *Archives of Internal Medicine*, November 1, 1940.

Abstracts from Medical Literature.

DERMATOLOGY.

Cheilitis and Dermatitis from Tooth Paste.

L. G. BERNHAUER (*Archives of Dermatology and Syphilology*, June, 1940) reports two cases of cheilitis and dermatitis from tooth paste. In each case the tooth paste gave positive results to a patch test with the patients and negative results with the controls. In the second case, in which the patient was sensitive to "Kolynos", the offending drug was found to be thymol, one of the ingredients of "Kolynos" tooth paste.

Sudden Death in Infantile Eczema.

J. H. DAVIES (*The British Journal of Dermatology and Syphilis*, June, 1940) reports the death in hospital of three consecutive patients suffering from infantile eczema. The circumstances of a typical case are the following. The accident is peculiar to infants with true infantile eczema and occurs during the first year of life. Undernourished infants are not so susceptible as large, fat, overnourished infants with a tendency to rickets. Most of the cases occur in the spring. Nearly all, if not all, deaths take place in hospital or away from the mother, during the first few days of the child's stay in hospital. The onset is heralded by improvement of the eczema. Autopsy never seems to disclose a sufficient cause of death. Death may be quite sudden and unobserved by the child's attendants. As a rule, however, a few hours elapse between the first symptom and the final outcome. The only premonitory sign may be the rapid healing or the sudden and unexplained improvement of the eczema. The first symptoms noticed are restlessness and pallor with or without cyanosis. Dyspnœa soon becomes obvious and may be intense. The infant becomes collapsed and unconscious, the lips become blue, the skin livid and the extremities cold. Temperature rises rapidly to 103° F. or more. Various theories by previous observers as to the cause of this phenomenon are quoted. To a great extent the clinical picture resembles anaphylactic shock. Anaphylaxis would fit in with the rapidity of onset, the pallor, the syncope, the dyspnœa and the convulsions. The author refers to the possibility that the cause of this accident may be psychological. He concludes that there is no room for disbelief in the actuality of "eczema death", that an indispensable factor in its production is hospitalization or at least separation from the mother, and that there is evidence that the prompt administration of adrenaline can be of value in treatment.

Early Syphilitic Lesions Mistaken for Dermatophytosis.

E. W. THOMAS AND S. M. BLUEFARB (*Archives of Dermatology and Syphilology*, July, 1940) state that amongst the commonest lesions of secondary syphilis are moist papules, which are usually found about the genitals and anus. When fused in plaques, they form the familiar *condylomata lata* of secondary syphilis and it has long been known that their growth is favoured by the moisture, heat and friction of opposing surfaces. One of the less common places for such lesions, and one that may be overlooked and thus cause an error in diagnosis, is between the toes, especially if the patient has an associated dermatophytosis. In the authors' experience it is not uncommon to see patients who have attended clinics and have been treated for dermatophytosis when an associated syphilitic process is present. The moist boggy skin with macerated opposing surfaces that characterizes fungous infections between the toes offers an ideal place for the localization of the *Spirochæta pallida* and the formation of moist papules, which in some cases present the typical picture of *condylomata lata*. The authors saw six cases of secondary syphilitic lesions between the toes from September, 1938, to February, 1940, and in five of these treatment had been given for varying lengths of time for stubborn dermatophytosis. When the lesions failed to respond to the usual treatments the patients were referred to the dermatological ward, where the presence of early syphilis was diagnosed and confirmed in every case by the finding of *Spirochæta pallida* on dark-field illumination. In each instance spirochætes were found in serum taken from lesions between the toes. Dark-field illumination of fluids taken from the lesions of dermatophytosis in patients without early syphilis have never shown similar spirochætes in their experience. All the patients in whom *Spirochæta pallida* was found had strongly positive Wassermann reactions of the blood. Moist papules between the toes may persist, like lesions in the throat, after all other cutaneous lesions have disappeared.

Sulphonamides in Lupus Erythematosus.

H. W. BARBER (*The Lancet*, March 30, 1940) states that most persons suffering from *lupus erythematosus* suffer severe reactions when sulphonamides are administered to them even in small doses. The symptoms produced comprise fever, scarlatiniform eruptions, tender lymphoglandular swellings, focal reactions in and transitory erythema around the patches, and temporary exacerbation of fibrositis or arthritis if these conditions should be present. In most patients, if administration of the drug can be continued, the lupus improves or disappears, as do any coincident symptoms of fibrositis or arthritis.

Histaminase in the Treatment of Allergic Dermatoses.

L. C. GOLDBERG (*The Journal of the American Medical Association*, August 10, 1940) reviews the literature on histaminase, an enzyme which inactivates histamine by oxidation, and suggests its use in allergic cutaneous disorders. Histamine may be obtained from the liver, lungs, skin and other body tissues in a pure form under aseptic conditions. It is preformed in the cell in a slightly inactive form and is readily released into the surrounding area. This release does not depend entirely on the destruction of the cell, but may occur when the cells receive stimulation of a certain intensity, and the magnitude of this stimulation is dependent on the allergic irritability of the individual cells. Histaminase is a compound of physiological albuminoid substance obtained from the intestinal mucous membrane. It is a loose white powder, which is stable and dissolves in water. The unit of histaminase is the amount necessary to inactivate one milligramme of histamine during twenty-four hours' incubation at 37° C. in a phosphate buffer solution at a pH of 7. Chemically it is diamine-oxydase. The author gives the results of treatment in 35 cases of allergic skin diseases; in 25 medication was given *per os* and in 10 by injection. One patient with papular urticaria was cured, the condition of three patients suffering from atopic dermatitis improved, of three patients with allergic eczema two manifested improvement and one did not. Of thirteen patients with urticaria (chronic) eight were clinically cured and five showed improvement. All three patients with idiopathic pruritus showed slight improvement. All the above patients were treated by tablets given by mouth. Ten patients with chronic urticaria were treated by injection and manifested improvement. The injections of histaminase were given every other day. The first two cubic centimetres caused fever up to 102° F., general malaise and pain at the site of the injection. After the second and third injections mild rises of temperature occurred and thereafter very little reaction. It is suggested that the histaminase may give the epidermal structures relief from histamine excitation and help them to build a tolerance to histamine, although there is no established fact that histamine is the causative agent of any pathological condition. Nevertheless evidence has been presented showing that histaminase oxidizes histamine in the body, and on this basis its therapeutic use in the treatment of allergic dermatoses may be justifiable.

Exfoliative Dermatitis due to Naphthalene.

S. J. FANBURG (*Archives of Dermatology and Syphilology*, July, 1940) reports a case of exfoliative dermatitis due to naphthalene, the eruption resembling *mycosis fungoides*. The patient's dermatosis was similar to cases reported by Sultzberger and Garbe under the title "Distinctive Exudative Discoid and Lichenoid Chronic Dermatosis", and by Cannon under the title "Allergic Dermatitis Simulating Lymphoblastoma". While an allergic basis for the condition was surmised by Cannon, Sultzberger and Garbe could not find sufficient evidence to "consider this dermatosis a representative form of any atopic manifestation, although it may be some form of sensitization dermatosis". In his case the author was able to find an allergen, naphthalene, ordinarily used as mothballs and flakes in the patient's home, the elimination of which was responsible for the disappearance of the exfoliative dermatitis. The exfoliative dermatitis resembled *mycosis fungoides* so closely that several competent dermatologists had made that diagnosis clinically. The author comments that if the precipitating cause had not been determined the damage to the patient's reticulo-endothelial system could have been carried to the extent that true *mycosis fungoides* or possibly Hodgkin's disease might have developed owing to repeated insults to that system. The clinical course of the condition was characterized by periods of exacerbation and remission. The author was struck by the fact that the exacerbations usually followed the patient's return home, and the acute symptoms always subsided quickly on admission to hospital. One relapse occurred whilst the patient was in hospital.

This relapse followed contact with a pair of trousers which were brought from the patient's home to be worn by him in hospital. As seven years have elapsed without recurrence, the author thinks that *mycosis fungoides* can be excluded, and suggests that more careful allergic study be made in proved as well as suggestive cases of lymphoblastoma, especially with a history of remissions whilst the patient was in hospital.

UROLOGY.

Trend of Transurethral Resection.

HERMAN KRETSCHMER (*The Journal of the American Medical Association*, July 13, 1940) reviews the progress of perurethral treatment of prostatic obstruction during the past eight years, which is the period of its more or less general application by urologists. During the earlier part of the period many articles dealt with bad functional results and dangerous complications. However, with increasing knowledge of technique, surgeons have been able to avoid such complications, and therefore poor functional results have been obviated. The author states that most urologists are now agreed that all bladder neck obstructions except large adenomata and difficult carcinomata are best treated by the urethral route. The author himself has found the possibility of applying endoscopic resection to be even greater, and in the last four years he has performed only one prostatectomy. He pleads for the application of the benign method of treatment in the very early stages of bladder neck obstruction, so that in future the classical secondary obstructive effects on the urinary tract may not be seen, and patients will be spared much ill health. Preparation for resection should be carefully carried out, though cases of greater risk can be attempted by this method than by prostatectomy. Preliminary cystostomy is found to be seldom necessary when experience is mature. The question of vesical diverticula as a complication of prostatic obstruction is important. When the obstruction is removed, nothing need be done for small diverticula. The same applies to moderate diverticula, unless the sac is pressing on and obstructing the corresponding ureter, when diverticulectomy is called for. Very large diverticula usually retain urine continually and cause permanent severe infection of the urine. The author in such cases removes the diverticulum first and later performs the resection. Larger amounts of adenomatous tissue are now being removed at resection than formerly, in some cases all the prostatic adenoma being resected. Owing to improved technique, hæmorrhage, early or secondary, has ceased to be a serious problem and is easily dealt with through the resectoscope itself, so that the suprapubic cystotomies of the early days for this urgent purpose are now seldom necessary.

Sulphamethylthiazol Therapy.

G. CARROLL, L. KAPPEL AND B. LEWIS (*Urologic and Cutaneous Review*, July, 1940) present a report of an experimental study of a new chemical product, sulphamethylthiazol ($C_{10}H_{11}N_3O_2S_2$), which as yet is not commercially available. Owing to the appearance of peripheral neuritis in some of the patients under treatment, the drug has been restricted to experimental use only until this matter has been cleared up. This potent sulphanilamide derivative seems to exert a powerful action against virulent strains of *Staphylococcus aureus*, both in laboratory animals and in human patients. This is very useful, since staphylococci are generally resistant to both sulphanilamide and sulphapyridine. Animal experimental work, moreover, has shown that this new drug is effective against the pneumococcus, the *Streptococcus hæmolyticus*, the gonococcus, and many other organisms. This drug is definitely superior to all other agents in cases of staphylococcal septicæmia. The authors have treated some fifty patients with perinephritic abscess, cortical renal abscess, calculous pyelitis, furunculosis and osteomyelitis, and all the patients recovered. The drug is a white, tasteless, crystalline powder, and is administered by mouth in capsules or tablets of 0·5 gramme weight. The sodium salt, being quite soluble in water, is suitable for intravenous use. Large doses are given initially, such as 5·0 grammes to begin with, followed by 1·5 grammes every four hours for two days. After that, one gramme every four hours is usually sufficient to maintain an adequate blood concentration level. The absence of toxic symptoms was quite gratifying. Nausea was sometimes observed after the initial large dose, but this disappeared within a few hours. Drowsiness sometimes occurred, but disappeared when the drug was discontinued. No leucopenia, agranulocytosis or other serious blood disorder was observed.

Carcinoma of the Penis.

P. A. NELSON (*Urologic and Cutaneous Review*, July, 1940) considers that cancer of the penis is a preventible disease. Circumcision in early infancy offers practical prophylaxis. Small cancers less than 2·5 centimetres in diameter are controlled by radiation therapy or by conservative amputation. Radical amputation and block dissection are seldom necessary. When inguinal gland metastases are present, a two-stage operation should be performed. The conservative amputation is followed in four to six weeks by inguinal dissection.

Renal Calculi.

FURTHER studies in the formation of renal calculi have been carried out by E. C. Rosenow, junior (*The Journal of Urology*, July, 1940). The material was derived from 239 consecutive autopsies and the renal papillæ were studied in each case. Plaques of calcareous material were found in the renal papillæ in 22·2% of all specimens examined; calculi were found in 5·4%, and they were adherent to the papillæ in five cases (1%). Twenty-four control sections of papillæ without any macroscopically visible plaques revealed microscopic intramedullary calcification in four. Age, sex, arteriosclerosis and necropsy diagnosis were not important factors.

Carcinoma of the Prostate.

R. W. BARNES (*The Journal of Urology*, August, 1940) compares the results achieved by various methods of treatment in prostatic carcinoma. Endoscopic resection of the bladder neck is shown to be the best method of dealing with obstruction to the urethra. Prior to the introduction of this technique cystostomy was the most successful. The author also finds that longevity is promoted by deep therapy inasmuch as his patients who have had X-ray in addition to other methods of treatment have a longer survival period than those who have had no deep therapy. He does not advocate deep therapy alone.

Site of Action of Sulphapyridine.

ALTHOUGH most authorities believe that sulphapyridine exercises its bacteriostatic effect in the tissues, it has been suggested that its success in urological conditions is due to the presence of the drug in the urine. R. M. Nesbit (*The Journal of Urology*, August, 1940) describes a case in which gonorrhœal urethritis, prostatitis and epididymitis were successfully treated by 23 grammes of sulphapyridine although the patient had previously had a bilateral uretero-sigmoidostomy.

Bilateral Perinephric Abscesses.

THE occurrence of bilateral perinephric abscesses is very rare. L. F. R. MOLINA (*The Journal of Urology*, July, 1940) records such a condition in a man of twenty-six. During the drainage of the abscess on the left side a similar condition was found to be present on the right and also required drainage. The presence of bilateral pyelonephritis was proved by pyelography, and in both cases an extension to the renal parenchyma had apparently occurred from the upper calyces and subsequently ruptured on the surface.

The Bladder in Carcinoma of the Cervix.

H. E. SCHMITZ AND P. A. NELSON (*Urologic and Cutaneous Review*, September, 1940) discuss the effects on the urinary bladder of both the disease itself (carcinoma of the cervix) and the application of radium to the cervix. There is a close lymphatic connexion between the bladder and the cervix; thus, chronic endocervicitis is often responsible for a mild trigonitis. In the same way, an early cancer of the cervix may spread to the bladder floor, first as œdema, later as a small ulcer or nodule. Routine cystoscopy should therefore be done in all cases. Obstruction to the bladder outflow may result from invasion of the vesico-vaginal septum, causing a fixed elevation of the bladder floor, or by infiltration of the urethra. As regards the application of radium, it is to be remembered that the bladder mucosa is much more sensitive to radium than muscle or connective tissue. Primary reaction in the bladder, occurring within twenty-four hours, is simply an erythema, and is insignificant and inconstant. Secondary radiation effects may be seen at the end of one month in the form of severe congestion or even acute inflammation with painful symptoms. No ulceration is present. The changes are limited to the posterior wall. The most important of the destructive effects of radium occur later, as a tertiary or delayed reaction, which may not reveal itself for years. The lesion is the result of obliterative endarteritis, with consequent atrophy. A white, avascular central area is seen on the bladder wall surrounded by a zone of dilated vessels. Hæmorrhage is prone to occur with phosphatic deposition and sepsis. The ulceration may spread and cause vesico-vaginal fistula. It is important that correct diagnosis of the tertiary condition should be properly made, otherwise it is prone to be looked on as a cancer recurrence.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XXXII.

PROGNOSIS IN MENTAL DISORDER.

THE days of the defeatist attitude in regard to psychiatry are past, with the chains and dungeons and sight-seeing crowds of the mental hospitals of a century ago; but it behoves us to exercise due caution and to avoid the error of hastily accepting any new treatment as the panacea for all mental ills. Only too often some enthusiastic proponent of a new form of therapy rushes into print with a laudatory paper on the results achieved in half a dozen cases. Others may be stampeded into believing that at last the problems of the psychoses are solved, and much valuable time and effort may be wasted and the hopes of relatives, to whom a favourable prognosis has been given, once more be dashed. Enthusiasm is to be admired and encouraged, but not when it overrides judgement or is precipitate and ill-timed.

During the last few years certain forms of treatment have been used with outstanding success; they have made the outlook much more hopeful in conditions which were formerly regarded as incurable. The two psychoses in which the most benefit has been derived from these advances are general paralysis of the insane and schizophrenia. In both hopeless dementia was the ultimate outcome before the advent of malaria and electropyrexia in the treatment of the former and of "Cardiazol" and insulin shock therapy in the latter. Many patients who would formerly have been regarded as suitable only for the "chronic" wards of an institution are now being restored to a life of economic usefulness. In both instances it is somewhat difficult to determine how the result is achieved. How an artificially produced pyrexia can cure gross mental deterioration, and an induced convulsion degeneration of character and personality, remain obscure. Perhaps we should be grateful for the treatment without inquiring too closely into the means by which the results are obtained. The advantages of malaria are undoubted; but it still remains to be seen whether convulsion therapy produces only a remission or a permanent cure. It appears to be of most value in the slowly disintegrating personality, and least in the paranoid type of schizophrenia. It also has its place in the treatment of depressive states, in which frequently considerable benefit results from a course of injections. Sometimes patients with manic reaction types respond too, but rarely those with confusional conditions and other psychoses. In a branch of medicine which is hardly out of its swaddling clothes, such steps forward are very encouraging and augur well for the future.

The adoption of the above methods of treatment has the further advantage of imparting a desirable stimulus to research in the physiological accompaniments of behaviour, an aspect that perhaps lagged behind psychopathological observation. Of course one must not lose sight of the psychogenic origin of many mental disorders in the search for a physical basis of insanity. The ruthless eradication of foci of sepsis as a cure for the psychoses had a brief vogue before passing into the limbo of things forgotten. An abscessed tooth, a hypertrophied tonsil, a chronically infected appendix or even a lacerated cervix was regarded as sufficient for a hopeful prognosis, because it had been forgotten that normal people suffered from these but developed no mental instability; this fact proved that there must be some other factor to which serious consideration should be given.

The question of prognosis is inextricably bound up with our knowledge of the ætiology of the particular disorder. Knowing the factors that gave rise to it, we can with greater assurance predict the outcome. Unfortunately, in many psychoses we are still in the dark concerning this aspect, and opinions can be given only as the result of experience with similar cases and their probable outcome. We are on safer ground when we are dealing with the toxic psychoses, the removal or elimination of the toxic element being the main concern of the psychiatrist. The facility with which this can be done shortens the course of the illness and makes the outlook correspondingly favourable. Vaccine therapy and such agents as vitamin B_1, in the case of alcoholic psychoses, are of inestimable value and serve to emphasize the importance of cooperation between the pathologist and the psychiatrist.

In the case of the dementias, primary or secondary, an unfavourable prognosis must obviously be given. The same applies to mental defect. In some of the involutional states one should be guarded in giving an opinion, for they are often protracted, lasting many months, and frequently intercurrent disease leads to a fatal termination. In most other conditions we can offer some straws of comfort for the relatives to clutch. In schizophrenia, which was formerly regarded as hopeless, recoveries are claimed for 40% of patients treated with "Cardiazol", and it has been stated that those who received injections within six months from the first manifestation of the disease have an 80% to 90% prospect of cure. Even if the hyperbole of the impulsive enthusiast is discountcd, the results are impressive. It still remains to be seen, however, whether there is complete and permanent recovery or merely a remission for a variable period. Some supporters of the physiogenic theory of the origin of mental disorders have seized upon these results as proof that their arguments are correct, maintaining that a condition which is cured by a physical agent must *ipso facto* have a physical cause. This is a strange inverted form of reasoning, which, however, is not solely confined to psychiatrists.

In the depressive states the ultimate prognosis is good, although progress may be slow and the condition may last for several months. Manic states are usually shorter in duration because of response to sedatives and such treatment as prolonged narcosis and hydrotherapy. In both there is a tendency to recurrence. It may be safely said that the earlier in life the onset, the greater is the likelihood of subsequent attacks with a possibility of chronicity eventually, and finally secondary dementia.

In confusional conditions due to some form of toxæmia a recovery rate of at least 70% may confidently be expected, provided that exhaustion can be prevented and the standard of general health raised. A small number of patients suffer a lasting impairment of intellect, especially of memory. Of these states the puerperal psychoses are usually of the longest duration. Confusion which has no apparent physical basis, unless associated with some other psychosis, has a favourable prognosis.

Although psychiatry has made rapid strides in the past thirty years, and one is justified in being more optimistic about the outcome, it is interesting to note that the recovery rate has steadily dropped since 1905, according to the latest report of the Inspector-General of Mental Hospitals of New South Wales. In this year 42·61% of persons admitted to mental hospitals were discharged, recovered, while in 1939 the rate was only 28·08%. There are two possible explanations of this. One is that the responsible officers are more conservative in their estimate of what constitutes recovery, and the other that many more mental defectives and more patients with organic dementia are being admitted to hospital because of the activities of the Child Welfare Department and the appreciation of relatives of the need for care and supervision of elderly patients. The percentage of those discharged as relieved or improved has risen from 5·55% to 14·54% in the same period, making the totals 48·16% and 42·62% respectively; these offer less contrast.

In paranoid conditions one should be very cautious in offering an opinion, as these patients often learn to suppress their delusions of persecution, realizing that they are not acceptable to others; but the delusions may still persist and make the patients a source of danger to others once they are free from institutional supervision. Early certification is always desirable, and the outlook is uniformly poor.

In the psychoneuroses one can reasonably expect to achieve satisfactory results with patience and care. It is necessary to make a detailed and comprehensive investigation of all possible factors—psychological, environmental and physical. In these days of broadcast talks by psychologists and popular books on the mechanisms of the mind, the patient cannot be fobbed off with a pat on the back, an encouraging word and a bottle of bromide mixture. Suggestion, deliberate or otherwise, used in this manner is doomed to failure now that the patient has become psyche-conscious. The solution of the problem which the patient has found to be beyond her capacity has now become the duty of the physician. Fortunately, in most instances this is not difficult. One can always give a favourable prognosis in these cases, although sometimes a harassed psychiatrist may wonder if obsessive states should not be included among the psychoses, so resistant are they to treatment in many instances. The only thing which distinguishes an obsession from a delusion very often is the fact that the patient realizes its absurdity and the influence it has on his actions. Neurasthenia may also persist for a long time after the apparent cause has been eliminated or dealt with, and the same frequently applies to hysteria; but in all we can confidently predict ultimate recovery, although the underlying neuropathic predisposition makes further attacks likely under stress and anxiety.

One is justified in both the psychoses and the neuroses in being more optimistic than formerly. In the case of the psychoses, better equipped mental hospitals, larger staffs, modern forms of treatment and intensive research into the ætiology of mental disorders, have all contributed to early and complete recovery, although it is not apparently reflected in the figures already quoted. In the neuroses the taking of a comprehensive history, the careful elucidation of the patient's problems, an investigation of the home situation, and the judicious application of the appropriate theory of psychopathology to the solution of the difficulty will serve to help the patient to make a satisfactory adjustment to life.

Just as there are many physical diseases which cannot and never will be cured, so there must be mental disorders which nothing can affect. It has been proved that conditions which had been regarded as beyond help can be treated and cured; and perhaps some day a psychiatric Harvey or Lister may come along and point the way to the restoration to sanity of those patients who still resist our best efforts. The effects of "Cardiazol" suggest that this may not be just an idle thought, of the sort to which psychiatrists may be unduly prone.

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British Medical Association News.

SCIENTIFIC.

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

THE second annual general and scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on October 10 and 11, 1940. In the absence of DR. JAMES FLYNN, the President, who was on active service abroad, and of DR. CLAUDE MORLET, the Vice-President, the Council invited DR. ARNOLD LANCE to act as Chairman.

Those present included five members from Victoria, three from Tasmania, two from Queensland, and thirty from New South Wales.

Chairman's Address.

DR. ARNOLD LANCE expressed the admiration and appreciation felt by members for the contribution their President was making to the cause of Empire and of civilization. Dr. Lance then extended greetings to members from other States, and recorded the thanks of the society of Dr. Mervyn Archdall for his editing of the first issue of the society's transactions. He then read messages of goodwill from Sir James Barrett, Dr. Claude Morlet, Sir Henry Newland, and Mr. Arthur Sinclair of Edinburgh.

Dr. Lance recalled the inauguration of the society in the same hall two and a half years earlier, and reviewed its work and aims. He deplored the interruption, mainly as a result of the war, of plans which had been made for an investigation of blindness in Australia, and he urged members to follow in the footsteps of the truly great, whose guiding principle was ever that of service to their fellow men. Stressing the importance of the health of the community as a primary aim of any system of government, Dr. Lance predicted that medicine would in the future raise a louder voice in the legislature; he commended the New South Wales Branch of the British Medical Association for its initiation of broadcast health talks. In conclusion, Dr. Lance reminded members of the limitations of ophthalmological knowledge, and expressed the hope that by its meetings and deliberations the society might make at least a small addition to the sum of medical wisdom.

Society Business and Dinner.

The remainder of the morning was devoted to the business of the annual general meeting of the society. In the afternoon the scientific meeting began, and in the evening a dinner was held in the banqueting room of the Royal Automobile Club of Australia. The scientific meeting was resumed on October 11, the proceedings concluding at 6 p.m.

Air Force Eye Standards and Examination Procedure.

WING-COMMANDER W. D. COUNSELL (Hobart) read a paper on the subject of eye standards and examination procedure for the Air Force. He described in detail the many special visual requirements of pilots, gunners and bombers. Pilots should have reasonable vision, but in addition they had to have effective binocular depth perception if they were to make safe landings with consistency. Gunners and bombers, on the other hand, had to have very good direct vision; other factors were not so important for them as for pilots. Wing-Commander Counsell described in detail the factors necessary for effective binocular depth perception, paying particular attention to the problem of heterophorias. He analysed case records of several well-known cricketers for their bearings on the vision and muscle balance required for pilots. In conclusion, Wing-Commander Counsell emphasized the importance ophthalmologically of the present time, when many new problems were being presented for solution.

Some Aspects of Visual Fusion in Peace and War.

In the absence of DR. J. RINGLAND ANDERSON (Melbourne), his paper was read by DR. D'ARCY WILLIAMS (Sydney). Dr. Anderson's paper dealt with the many factors necessary for true orthophoria, and also with the necessity for air pilots to have an ample range and speed of accommodation, a complete range of binocular movements, good fusion and full amplitude of stereoscopic vision. It was pointed out that in the history of the human race the emergence of fusion and stereoscopic vision had ever been a survival factor; these faculties were no less so in view of the present combat.

Visual Complications of High-Speed Flying.

In the absence of DR. CONRAD BLAKEMORE (Sydney), his paper was read by DR. ARTHUR D'OMBRAIN (Newcastle). The paper investigated the phenomenon known as "blackout", a sudden, transient blindness occurring at certain speeds when the pilot in his plane was traversing a curved path. "Blackout" occurred if sufficient centrifugal force was exerted for a long enough time, and could be explained by the postulation of a condition of retinal anoxæmia, since the retina required for function about four times the amount of oxygen of any other tissue.

An Ophthalmologist in the Great War.

DR. R. GRANVILLE WADDY (Sydney) read a paper dealing with the undesirability of putting a specialist to work other than that for which he had been trained. Dr. Waddy mentioned the variety of work that he had been set to do during the Great War; some of it was ophthalmic, but much of it was work for a regimental or transport medical officer. He made suggestions for the more efficient testing and recording of visual acuity on enlistment, for the recording of the prescription of glasses in soldiers' paybooks, and for the making of careful notes about cases encountered on active service, so that repatriation officers might in due course have adequate information upon which to form a judgement as to the extent to which war service had caused or aggravated disabilities.

Blindness in Tasmania.

DR. J. BRUCE HAMILTON (Hobart) referred to the inability of the National Health and Medical Research Council to accede to the request of the society that an annual sum of £2,000 should be expended for three years in a survey of the blind in Australia. Dr. Hamilton pointed out that it was probable that such an expenditure would save the Commonwealth £25,000 *per annum* for twenty years. After having given details of the activities of the Tasmanian Prevention Committee, Dr. Hamilton discussed the successful sight-saving school in Hobart. (This was the subject of a leading article in this journal in the issue of February 24, 1940.) He presented an analysis of 121 cases of binocular blindness and 518 cases of monocular blindness found in a survey of 6,770 private patients. Dr. Hamilton made a plea for a complete survey of Australia, and pointed out the economical value of such a survey.

Applied Anatomy of the Subgeniculate Pathway.

DR. GILBERT PHILLIPS (Sydney) read a paper on the application of anatomical relations to the localization of intracranial tumours. He asked members whether they could give him any guidance in distinguishing inflammatory optic neuritis from papilloedema.

The Treatment and Prognosis of Sympathetic Ophthalmia.

In the absence of Dr. J. Ringland Anderson, his second paper was read by Dr. Arthur D'Ombrain. In this paper Dr. Anderson reported a case of sympathetic ophthalmia;

the patient was a boy, aged seven years, and the condition followed a wound of the left eye caused by a nail. The aspects of the subject discussed in the paper were: (i) the advice given to the doctor in charge of the patient; (ii) a basis for better advice; (iii) the patient's later history; (iv) prognosis and the relative merits of therapeutic measures. Several points were stressed, namely: (i) the site of the wound, which was near the equator; (ii) associated nasal sepsis; (iii) the absence of keratic precipitates in the injured eye; (iv) a second injury to the eye, from a fall against a pipe, before the onset of sympathetic ophthalmia. Dr. Anderson, in his review of the case, considered that the treatment was imperfect. The boy was not tested for sensitivity to tuberculin, diphtheria antitoxin or uveal pigment; no prophylactic treatment was given; removal of his eye was unduly delayed, and the subsequent therapeutic attack was too late and lacked concentrated effort.

Keratitis Sicca including Sjøgren's Syndrome.

Dr. Bruce Hamilton discussed several types of dry eye, particularly Sjøgren's syndrome. He prefaced his remarks by the statement that "nothing dries sooner than a woman's tears", and then went on to discuss Sjøgren's syndrome, the symptoms of which were dry eye with keratitis, idiopathic swelling of the salivary glands with dry mouth and anhydrosis; it occurred most commonly in women at the menopause and was accompanied by articular changes. Dr. Hamilton distinguished two types of corneal dryness—one in which diminished lachrymal secretion produced a secondary keratitis, and one in which the primary condition was a vesicular or filamentary keratitis. The former type was amenable to treatment by occlusion of the punctum and the use of artificial tears. The punctum and portion of the canaliculi should be obliterated by electro-coagulation, not merely cauterized. Artificial tears consisted of a 10% solution of blood serum in Ringer's solution, with the addition of one grain of chloretone per ounce as a preservative. Tests for dry eyes were carried out by means of strips of filter paper 25 millimetres long and five millimetres wide; they were bent over at one end and hooked over the lower puncta. In five minutes at least 15 millimetres of the strip should be saturated, or the eye was abnormally dry. Dr. Hamilton quoted cases to illustrate the success of this form of treatment.

Krukenberg's Spindle.

Dr. Granville Waddy described the Krukenberg-Axenfeld spindle, a vertical, fusiform, axial pigmentation of the posterior surface of the cornea. He said that the condition was essentially a melanosis of the epithelium. It was to be regarded as an indication of generalized pigmentary degeneration; vision was little affected by it. Dr. Waddy also dealt with factors governing the circulation of intraocular fluids, and showed the relationship between the intraocular fluid currents and the deposition of pigmentary and other keratic precipitates.

The Treatment of Trachoma with Sulphanilamide.

DR. CLIFFORD COLVIN (Orange) reported the treatment with sulphanilamide of 40 patients suffering from trachoma, and gave details of two of the cases. The drug was administered in doses of seven grains three times a day for a week, and after a week's spell, if necessary, the treatment was repeated for another week. Dr. Colvin considered that sulphanilamide was useful in trachoma and appeared to affect directly the virus or causative organism. However, frequently there was a residual granular condition that persisted; therefore local treatment to the lids was given as well.

Ionization.

DR. E. TEMPLE SMITH (Sydney) read a paper on ionization and gave a demonstration of zinc ionization of the lids. He said that anions such as atropine were attracted to the positive pole, and the solution was therefore applied from the negative pole. Zinc was a kation, and was applied from the positive pole. Dr. Temple Smith said that in squamous blepharitis a dose of one or two milliamperes applied for two and a half minutes usually cured the condition, and did not need to be repeated. In ulcerative blepharitis it was not so successful. In corneal ulceration the dosage should be 0·5 milliampere applied for thirty seconds, and the method should not be forgotten in perforating wounds, for it might save the eye from deep infection.

The Treatment of Pterygia.

DR. A. L. NORTH (Sydney) discussed pterygia. He said that the ætiology of pterygium and that of pinguecula were similar, namely, a degeneration of tissue that was exposed to wind and dust. Though the two conditions could occur quite independently, pinguecula predisposed to pterygium by interfering with the nutrition of the cornea at that spot. To prevent a recurrence, healthy conjunctiva should be drawn down so that it was opposite the palpebral fissure. For the treatment of recurrence, Dr. North described a method of inserting a mucous graft, or preferably a skin graft, at the limbus. The tiny strip was tucked under the overlapping conjunctiva at each end of the graft; no sutures were necessary. Dr. North showed a patient on whom the procedure had been successfully carried out.

The Lexer Operation for Ptosis.

DR. T. M. ARMSTRONG (Sydney) described the Lexer operation for ptosis and showed a photograph of a patient with congenital ptosis who had been successfully treated by this method. Two strips of *fascia lata*, three millimetres broad, were used to connect the frontalis to points three millimetres above the lid margin; they were placed in subcutaneous tunnels runing from the junctions of the middle and outer thirds and the middle and inner thirds to the lid margin. The condition was undercorrected, as the fascia contracted during the following seven weeks. No special precautions were necessary to protect the cornea.

Some Aspects of Glaucoma.

Dr. Temple Smith discussed tonometry, scotometry and operative methods in glaucoma. He considered that the Schiotz tonometer was the only one of real value, and he described the exact technique of its application to the cornea. Dr. Temple Smith then demonstrated his circular revolving Bjerrum's screen. It was considered that this Elliott scotometer had several advantages over other designs. Finally, Dr. Temple Smith gave his reasons for preferring the La Grange operation for chronic glaucoma, maintaining that it provided a more real drainage than the operation of trephining. He said that he relieved acute glaucoma by scleral puncture, and when the condition of the eye had become "quiet" he performed the La Grange operation.

An Analysis of Sixty-Five Cases of Glaucoma.

DR. N. M. MACINDOE (Sydney) analysed the operative results obtained at the Royal Prince Alfred Hospital in 65 cases—one of buphthalmos, 14 of secondary glaucoma, 11 of primary acute glaucoma and 39 of primary chronic glaucoma. As a result of his analysis, Dr. Macindoe came to the following conclusions: (i) The prognosis when visual acuity was low and the visual fields were small was very bad; therefore operation (trephining) should be performed early. (ii) The prognosis in glaucoma following the extraction of a cataract was bad; trephining or limbus section held out some hope. (iii) The prognosis in acute traumatic glaucoma was very bad. (iv) Iridectomy was much inferior to trephining in acute glaucoma. (v) In chronic glaucoma which was unrelieved by the use of eserine and in which operation was inadvisable, the intravenous use of hypertonic saline solution might be useful. Finally, Dr. Macindoe asked whether anyone could suggest a way to induce opticians and general practitioners to recognize glaucoma in its early stages, before the visual fields had become tubular and visual acuity as low 6/60.

The Berger Rhythm.

Dr. Gilbert Phillips described the electrical changes in the human brain known as the Berger rhythm. He said that electrical action potentials, which occurred spontaneously in the human brain, were now able to be measured and recorded graphically. These electrical changes, first described by Berger in 1929, were rhythmical in nature, occurring with a periodicity of ten per second; Dr. Phillips named them the "α" rhythm, but this was now generally identified as the Berger rhythm. Other rhythms had been described; but for ophthalmologists the Berger rhythm claimed chief attention, for it had been found particularly susceptible to inhibition by visual stimuli. The rhythm appeared to be best developed in cortical area XIX, on the lateral aspect of the occipital lobe. In any one individual the rhythm was constant in frequency and magnitude, and might be regarded as a cerebral index of personal identification. The Berger or "α" rhythm was first modified and then abolished in sleep, being replaced by a "δ" rhythm of different magnitude and frequency. Visual stimuli produced modification or even disappearance of the rhythm, and studies had been made of the relation between the fusion frequency of flicker and the resultant cortical action potentials. Electroencephalography had also been used in the study of the effect of disease processes on the electrical activity of the brain, particularly in epilepsy. In some cases it was even possible to foretell the subsequent occurrence of a par-

ticular form of epilepsy in an apparently healthy individual. Neoplasms influenced the rhythm of the nearby cortex. A complete electro-encephalographic unit had been ordered for the Royal Prince Alfred Hospital.

A MEETING of the Victorian Branch of the British Medical Association was held on September 10, 1940, at Saint Vincent's Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Parts of this report appeared in the issues of December 28, 1940, and January 11, 1941.

Parkinsonism.

DR. JOHN F. WILLIAMS and P. GUY REYNOLDS showed a male patient, aged fifty-nine years, suffering from early senile Parkinsonism. His main complaint over two years had been of pain down the left side. This was associated with lack of swing in the left arm when he was walking, and this arm had felt colder than the right. There was nothing suggestive of encephalitis in the previous history, and routine examination of the central nervous system revealed no change in the reflexes; but a lack of swing and slight rigidity in the left arm were noted, together with some slight left-sided facial immobility. His systolic blood pressure was 180 and his diastolic pressure 100 millimetres of mercury. Dr. Williams and Dr. Reynolds referred to the difficulty of early diagnosis and emphasized the frequency with which pains were misleading; the correct diagnosis was often missed on that account.

Dr. Williams and Dr. Reynolds then showed a male patient, aged forty-five years, who had come into their care because of pain in the upper cervical region, associated with inability to move quickly. His previous history was somewhat difficult to elicit, but at about the age of nineteen years he had had concussion, probably with intracranial hæmorrhage, and at some time shortly after this he had "meningitis". Following these illnesses he became dizzy, the pain in the neck commenced, and he also began to have fits in which he was reported to have injured himself. Some five years previously he had been transferred to the Receiving House as a voluntary boarder, as home care had been difficult; but he was detained there only for a short period.

On examination there was no sign of mental disturbance, though the patient's appearance was one of dulness; pronounced Parkinsonism, cogwheel rigidity, stooped posture and rigid facies were present, and there had been a lack of education in his early years. There was, however, no evidence of any delusions or hallucinations or anything suggestive of a schizophrenic psychosis. None of the "fits" had been seen until July, 1940, when he was seen in the out-patient department of the hospital in a condition of catatonia. During this attack he spoke slowly and somewhat indistinctly, could apparently make no voluntary movements of the limbs, and when placed in position retained this position indefinitely (*flexibilitas cerea*). During this attack, which lasted for over an hour, no change in reflexes was elicited. Further investigation of the history revealed that he had been having somewhat similar attacks over a long period; the phenomenon of *flexibilitas cerea* was well demonstrated, though he retained voluntary movements of all limbs.

Dr. Williams and Dr. Reynolds referred to the very unusual nature of this finding, and discussed the possible localization of the lesion responsible. Treatment with *Tinctura Stramonii* and benzedrine sulphate had led to some improvement, though there were still persistent complaints of pain which were regarded as due to the muscular rigidity. The possibility that the condition was traumatic in origin was also discussed.

The third patient shown by Dr. Williams and Dr. Reynolds was a woman, aged twenty-five years, who also had been referred to them on account of pain. This was in the leg and was associated with a tendency to drag the limb when walking; there was also a previous history of slowness of movement for ten years following on an illness characterized by excessive sleepiness some two years before. Further investigation revealed a seven years' history of attacks in which the eyes rotated upwards for periods up to one hour except for momentary periods when the eyes returned to their normal position.

Examination revealed a left-sided Parkinsonism, mask-like expression, and rather prominent eyes. The patient was treated with increasing doses of *Tinctura Stramonii*, and from 1938 onwards with benzedrine sulphate, with some relief from her oculogyric crises. Emphasis was placed on the scanty history of encephalitis in the majority of these cases, and also on the way in which oculogyric crises were often induced by emotional tension.

Muscular Dystrophy.

Dr. Williams and Dr. Reynolds then showed a male patient, aged thirty-five years, who had for eight years suffered from weakness in the thighs and back, which rendered him unable to stand up from a sitting position without support. He had, however, from school days onwards been clumsy in movement and games. On examination some hypertrophy of the calves was noted, with wasting of the thigh, buttock and shoulder girdle muscles. In addition there was a pronounced waddling gait of the type associated with pseudo-hypertrophic muscular dystrophy. He had been under observation for five years, and during that time there had been little or no change in his condition. Treatment had at one time been with 20 grammes of glycine a day. This was followed by 100 grains of gelatin a day for two years; but since 1936 he had been having half a grain of ephedrine twice a day.

The condition of the next patient shown by Dr. Williams and Dr. Reynolds contrasted with the chronic course; the patient was a girl, aged fifteen years, with a history of being quite capable until at least Christmas, 1939. Symptoms really were complained of only in March, 1940; some aching and weakness in the back occurred. In April the patient began falling in the street owing to loss of control of the knees but no loss of consciousness, while there had recently been some difficulty with reading and knitting, and also at times some difficulty with nasal speech, the tongue apparently seeming to swell. The past history was clear, except for an attack of left facial palsy at the age of ten years, lasting for some six weeks. There was nothing important in the family history, as in the previous case. Examination revealed board-like, somewhat hypertrophied calf muscles, weakness and wasting in the thigh, buttock and shoulder girdle muscles, together with a myopathic facies. Treatment with "Bemax" had been of short duration; but there had been quite pronounced deterioration in her condition in the few weeks she had been under observation.

Asthma Treated by Desensitization by the Nasal Method.

DR. GERALD DOYLE showed three patients suffering from asthma who had been treated by the intranasal method of desensitization. These three patients were selected from a group who had undergone this method of treatment, and their ages were fifty-six years, forty-five years and fifteen years respectively. They had had asthma intermittently over periods of fifteen, ten and four years respectively.

Dr. Doyle said that that method of desensitization had been applied to those subjects who did not give very strong reactions to the ordinary skin tests. The procedure adopted was to inject the vaccine into the mucosal layer of the nose, covering the septum and inferior and middle turbinate bones. The results so far had been very successful. Dr. Doyle in showing these patients did not intend to suggest that this method of desensitization would supplant all others; but in a series of 40 cases it had proved to be very successful. The patients shown had had no attacks since beginning the treatment; one patient had had two attacks and another a mild attack.

Microscopic Sections of Ophthalmic Interest.

DR. KEVIN O'DAY showed a number of microscopic sections. The first were sections of the normal optic disk and the *fovea centralis* which had been stained with hæmatoxylin and phloxine as well as with Mallory's connective tissue stain. The eye from which the sections were taken was normal; it had been removed by Dr. Leo Doyle whilst he was eradicating a malignant tumour of the face. The vision in the eye had been normal, and the fundus was normal; a pronounced physiological cup was visible in the centre of the optic disk. Dr. O'Day said that the sections demonstrated the anatomical structure of the physiological cup, the normal structure of the optic nerve entrance and the normal structure of the *fovea centralis* and surrounding tissues.

The next microscopic sections were sections of a whole eye in which papillœdema was demonstrated; the eye was from a patient suffering from a cerebral tumour. Sections of another whole eye demonstrating papillœdema were shown; the eye had been taken from a patient suffering from yeast abscess of the cerebrum. The swelling of the disk was more pronounced than in the previous case and was accompanied by a larger amount of hæmorrhage. A macular "fan" was observed ophthalmoscopically. Microscopic examination revealed pronounced swelling of the optic disk, congestion of the veins, perivascular exudation and hæmorrhage, cytoid degeneration of nerve fibres and subretinal exudation. Dr. O'Day said that the subretinal exudation was not a post-mortem artefact, and was probably responsible for the appearance of the macular "fan". Yeast cells were present in the subarachnoid space of the optic nerve.

Dr. O'Day then showed microscopic sections of the optic nerves, optic chiasma and left optic tract from a patient suffering from cerebral tumour associated with defects in the visual fields due to involvement of both papillo-macular bundles. The field defects were of the nerve-fibre bundle type usually associated with glaucoma. The tumour was so extensive as to be inoperable. At autopsy Dr. Morgan found that the roofs of both optic canals as well as the roofs of both orbits were greatly eroded. The finding of a glaucomatous type of field defect was difficult to explain, as it was usually assumed that the lesion in such cases was in the distal part of the nerve—actually at the optic disk itself. To preserve the normal anatomical relations, both globes with the optic nerves, chiasma and left optic tract attached, were embedded in celloidin. Sections were cut at regular intervals along the optic nerves, chiasma and left optic tract, and stained by Weigert's method. The degenerated papillo-macular bundle was well seen; in the posterior part of the optic nerves and in the chiasma it lay on the dorsal aspect. Dr. O'Day remarked that that finding was at variance with that of Uhtoff, Brouwer, Zeeman and Henschen, who placed the papillo-macular bundle in the centre of the posterior part of the nerve and in the same position in the chiasma. In this instance the dorsal position of the papillo-macular bundle explained the nature of the defects in the visual fields; the lesion was caused by direct pressure on the papillo-macular bundle.

Visual Fields in Fracture of the Skull.

Dr. O'Day then showed a female patient, aged twenty-one years, who three months previously had sustained concussion in a motor-car accident. At the time of the meeting she was complaining of a spot in front of her eyes. The vision in each eye was $^6/_6$ and the fundi were normal. In the right visual field the patient had an arcuate scotoma extending from the lower edge of the blind spot below fixation point to the horizontal raphe. A similar lesion was present in the left eye; but instead of extending to the "raphe" it "broke through" below to the nasal periphery. Both defects had sharply defined edges, and the scotomata were so absolute that the observer's hand could not be seen in them on the Bjerrum's screen at a distance of two metres. An X-ray examination revealed a fracture of the base of the skull involving both optic canals. Dr. O'Day remarked that in the light of the anatomical findings in the previous case it appeared that in the fracture involving the optic canals both papillo-macular bundles were injured as they lay on the dorsal aspect of the optic nerves in the optic canals.

Hydronephrosis.

DR. FRANK CAHILL showed a male patient, aged forty-six years, who had for years been subject to attacks of left-sided upper abdominal pain, not related to food, lasting about half an hour, and associated with retching of bile. He was for a period of months at times free from pain. His appetite was quite good apart from the attacks. His bowels were open quite regularly, and he had no symptoms of urinary infection.

On examination no abnormality was detected in the heart or lungs. Tenderness on deep pressure was elicited in the right side of the hypochondrium, and some generalized epigastric rigidity was present. There was some suspicion of a mass palpable in the right side of the hypochondrium, but it was not definite.

In view of the gastro-intestinal symptoms, an X-ray examination after a barium meal and a cholecystographic examination were made, with negative results; but in the films several irregular shadows in the region of the pole of the right kidney, suggestive of renal calculi, could be seen. A cystoscopic and a pyelographic examination were then made; the left kidney appeared normal in outline, the urine was sterile and dye appeared in good concentration in four minutes. On the other side the urine contained numerous pus cells and bacilli; the function was poor, dye appearing only after ten minutes, and the outline revealed a pelvic hydronephrosis, with calculi lying in the lower pole. In view of the condition found, it was considered that nephrectomy was the correct treatment, and the patient at the time of the meeting was awaiting admission to hospital for operation. Dr. Cahill said that the interest in the case was the complete absence of any symptoms suggestive of a renal lesion.

Dr. Cahill then showed a female patient, aged thirty-five years, who had had for twelve months a continuous aching pain in the right loin and right iliac fossa, much worse at some times than others; the severe attacks were associated with nausea and vomiting and lasted up to six hours. She had had an appendicectomy six months earlier, when it was discovered that the right ovary was also diseased. Since then the patient had been no better, and the pain had been worse during her menstrual periods. Her appetite was poor; the bowels were open regularly, but she had lost about seven pounds in weight in the last six months. She had an attack of pyelitis twelve months earlier, and another one month prior to the meeting; since then she had had persistent frequency of micturition, passing urine four or five times during the day and two or three times at night. She had had a tonsillectomy twelve months earlier.

On examination no abnormality was detected in the heart or lungs. There was a scar in the right lower paramedian area of the abdomen. Extreme tenderness was present in the right iliac fossa, and slight tenderness on the right side *per rectum* and in the right fornix. A catheter specimen of urine at that stage was normal.

The patient was seen again a week later; she reported feeling better, but had a temperature of 99·2° F. and a pulse rate of 100 per minute; tenderness was still present, more so in the right loin. A plain X-ray film revealed no abnormality in the renal areas, but a shadow was seen in the region of the right transverse process of the fourth lumbar vertebra; it was suggestive of a calcified gland, but was in the region of the ureter. A "shift" film with ureteric catheters in position showed the shadow to be apart from the ureter, and a pyelographic examination on both sides showed the left kidney to be normal and of good function, while the right kidney was ptosed and hydronephrotic, taking 30 cubic centimetres of sodium iodide. There appeared to be a definite kink at the uretero-pelvic junction, and the urine from the kidney was sterile; dye appeared in poor concentration in fifteen minutes, compared with three minutes in the left kidney. Apparently the pain complained of was due to a hydronephrosis, and the exacerbations were probably Dietl's crises. It was considered feasible to perform a nephropexy, and at operation a lobulated kidney was found and sutured to the posterior abdominal wall after the removal of most of the perirenal fat.

Wrist Injuries.

Dr. Cahill then showed a male patient, aged twenty-four years, a boiler-maker, who had fallen off his bicycle on May 11, 1940, and injured both wrists. An X-ray examination revealed a Bennett's fracture of the right thumb and an anterior dislocation of the left semilunar bone and fractured scaphoid, the proximal fragment being dislocated forwards with the semilunar bone. The dislocation and fractures were reduced under general anæsthesia and both wrists were placed in a plaster cast. On June 12, 1940, the plaster cast was removed from the left wrist and another X-ray examination was made. The proximal fragment of the scaphoid bone was definitely more dense than the rest of the carpus; the condition was suggestive of aseptic bone necrosis.

Removal of the fragment was advised. On July 19, 1940, the proximal fragment was excised under general anæsthesia and the wrist was replaced in a plaster cast. By this time the Bennett's fracture had united in good position and with good function. At the time of the meeting the patient had returned to his work of boiler-making with no disability, though limitation of about 15° in both volar and dorsal flexion was still present at the wrist joint. There was no pain, and the grip was equally as strong as on the other side.

Congenital Abnormalities.

Dr. Cahill then showed a male patient, aged seventy-two years, who on September 2, 1940, had complained of loss of a few pounds in weight in the last six months, some right-sided upper abdominal pain after meals, and a loss of appetite for meat. He had no previous history of indigestion of any form whatsoever.

On examination no abnormal physical signs were detected apart from some diminution of air entry at the base of the right lung, and kyphosis, senile in type. The possibility of a carcinoma of the stomach was considered, and a barium meal examination was carried out by Dr. H. M. Hewlett. It was found that the œsophagus was abnormally short, ending about the level of the fourth dorsal vertebra, and the whole of the stomach was supradiaphragmatic, lying apparently in the posterior mediastinal space. The stomach was also dilated and the pyloric extremity pointed towards the left; and occupying the œsophageal hiatus of the diaphragm was the second part of the duodenum, which apparently was directly continued into the jejunum. Peristaltic waves could be seen crossing the stomach on the screen, and there was no question of its being a grossly dilated œsophagus. Apparently there was a failure in elongation of the œsophagus in intrauterine life, and the stomach maintained its fœtal position, the diaphragm developing below it instead of above it. The condition up to date had caused no symptoms, which was all the more surprising since the patient had done laborious work all

his life. Dr. Cahill remarked that in the reported cases of this condition the patients usually suffered from dysphagia very early in their careers.

Secondary Deposits in Bone from an Epithelioma.

Dr. Cahill finally showed a male patient, aged thirty-four years, who had attended the hospital as an out-patient four years earlier, complaining of a small ulcer on the lower lip just to the left of the mid-line; the ulcer was excised with a large wedge of lip. He said that four years earlier he had bitten his lip and a small nodule appeared, which six months before had ulcerated. The pathological report was that the ulcer was an epithelioma. There was no evidence of any glandular involvement. For the six months prior to the meeting the patient had noticed a gradual enlargement of the left angle of the lower jaw, which had gradually been growing painful, and he could no longer open his mouth more than about one inch.

On examination a hard smooth enlargement of the angle of the jaw was found, extending outwards. It was definitely tender on external pressure. It was thought to be possibly an osteoclastoma; but as there was a thickening along the line of the upper cervical glands below the angle of the jaw, the possibility of a secondary malignant deposit was considered. The Wassermann test produced no reaction, the leucocytes numbered 8,800 per cubic millimetre, and on X-ray examination a sharply outlined bean-like defect was seen on the lower margin of the left mandible, just in front of the angle of the jaw; the radiologist suggested the possibility of tuberculosis. Another film, taken a month later, revealed definite progress in the destruction of the bone, which appeared like a secondary neoplastic involvement. It was considered that the correct treatment would be to insert radium needles in the region of the mass; but the ultimate prognosis appeared poor. Dr. Cahill said that the interest in the case was the rarity with which a secondary malignant deposit from an epithelioma involved bone in this manner.

Pernicious Anæmia.

DR. T. E. LOWE showed a number of patients suffering from pernicious anæmia. These patients had been under treatment for varying periods of up to eight years. The records of blood examinations of these patients showed that liver therapy given by mouth alone did not satisfactorily control the erythrocyte level nor the hæmoglobin content of the blood. Remissions and relapses were seen in the blood condition when the liver was given solely by mouth. The changes ran in cycles of approximately six months' duration, the number of erythrocytes varying from 5,000,000 to 3,500,000 cells per cubic millimetre. This suggested that liver therapy merely masked the remissions and relapses of the untreated disease, but did not adequately control them when it was given in the usual doses by mouth.

During the period when these patients were given liver extract of various types in adequate dosage parenterally, the relapses and remissions were completely controlled. This apparently was due to the reservoir action of the injected liver, which gave a supply of hæmatopoietic principle adequate to control the blood in both remissions and relapses.

Other important points in treatment were also illustrated. Most patients suffering from pernicious anæmia required iron salts in addition to liver to maintain a satisfactory hæmoglobin level. When the colour index fell below 1·0 iron should be given in addition to the liver. It was also apparent that repeated blood examinations at intervals of two to three months were essential if remissions and relapses were to be adequately followed and checked. It was also evident that the optimum levels of 5,000,000 erythrocytes per cubic millimetre and 100% hæmoglobin could easily be achieved and maintained by regular injection of liver extracts at intervals of two or four weeks or more, according to the individual patient, provided that iron salts were also given when necessary.

Correspondence.

MEDICAL ASPECTS OF CRIME.

SIR: Dr. Dark's reference to the medical aspects of crime is full of interest. I cannot rival him in detailed knowledge of criminals, but have sufficient information to make me realize that punishment and even execution are necessary, the latter, as Dean Inge put it, not for revenge or even example, but for the same reason that we destroy a dangerous animal.

Some years ago a man in a Melbourne gaol served an exemplary ten years' sentence for violence. The Inspector-General informed me that the police throughout Australia were warned that he would certainly commit murder if anything stood in his way. He was released and, I think, in eighteen months was sentenced to death and executed for murder in Queensland. Is it worth while to let such people loose?

Some time ago I visited an excellent Honour Prison at French Island, Victoria. There were about 30 prisoners (indeterminate) and four warders. Excellent food and accommodation were provided, wireless was installed and there was plenty of useful hard work. It did not look like a prison in any way. Yet every now and then a prisoner tried to escape, sometimes a few months before his freedom had been recommended and ordered. Why does such a one behave like this when all concerned desire to help him?

Again, why do many youths steal motor cars, use them and sometimes destroy them? Something may be done to make the road to betterment possible, but it is not easy. Nor is the road very definite from the medical or any point of view.

Again with sexual offences, especially against children, how can severe punishment be avoided? Are not these offenders abnormal?

At the Honour Prison the officer in charge informed me that one sexual offender whom I met, and who was doing excellent work, could not be released as it was almost certain he would commit another sexual offence.

My sympathies are in many respects with Dr. Dark, but my knowledge of prisons and criminals makes me realize that we have to walk warily. Poverty is not the cause of gross crime; crime arises from a determination to obtain goods without work and some of the criminals I have met are the most plausible and pleasant persons possible.

When men are dangerous there is no alternative. They must be shut up or destroyed. Dr. Dark, like so many other sincere investigators, seeks for the cause of the appearance.

With this in mind the American playground system came into being and all sorts of efforts have been made and are being made both in the United States of America and now in Australia. But it is difficult to get the necessary self-discipline with young people. What we are all trying to do is to make people conform to the rules of civilization. But one cannot evade the conclusion that a certain very limited number of men and women must be shut up for life or executed.

Yours, etc.,

103-105, Collins Street, Melbourne,
January 20, 1941.

JAMES W. BARRETT.

MEDICAL OFFICERS AND FULL-TIME HOME SERVICE.

SIR: I noticed in your issue of January 18, 1941, "Medical Officers required for full-time duty" (Home Service), Victoria.

I take it that these applicants will be men unfit for the Australian Imperial Force or over age. For such men the same conditions should apply as to members of field ambulances and other medical corps who get rank and only do an occasional fortnight.

Separation allowance is granted to their wives, which is not given to men over the age of enlistment who are willing and able to do all camp and hospital duties. I would like this matter taken up with the Military Authorities.

Yours, etc.,

"FULL TIMER."

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, as duly qualified medical practitioners:

Hoy, Ronald James, M.B., B.S., 1939 (Univ. Sydney), c.o. Mr. C. A. Hoy, 32 Marne Street, South Yarra, S.E.1, Victoria.

Stout, Alta Frances, M.B., Ch.B., 1935 (Univ. Edinburgh), c.o. National Bank of Australasia, Sydney.

Gray, John, M.Chir., 1929, M.D., 1936 (Univ. Cambridge), F.R.C.S. (England), 1928, 31, Boronia Road, Bellevue Hill.

Verso, Murray Linton, M.B., B.S., 1940 (Univ. Melbourne), District Hospital, Marrickville.
Lyth, Oliver, M.R.C.S. (England), 1933, L.R.C.P. (London), 1933, Narrandera.
Welch, Edwin Steuart, M.B., B.S., 1926 (Univ. Melbourne), Tumbarumba.
Brodsky, Alexander Gregory, M.B., 1940 (Univ. Sydney), c.o. New Medical School, University of Sydney, Sydney.
Lackey, Samuel, M.B., B.S., 1940 (Univ. Sydney), 35, Cavendish Street, Stanmore.
Owen, Morris David, M.B., B.S., 1940 (Univ. Sydney), Newcastle Hospital, Newcastle.
Berger, Pinkas, M.D., 1935 (Univ. Bratislava), 55, Temple Street, Stanmore. Registered in accordance with the provisions of Section 17a of the *Medical Practitioners Act*, 1938-1939.
Rubinstein, Kusiel, M.D., 1936 (Univ. Paris), District Hospital, Balmain. Registered in accordance with the provisions of Section 17a of the *Medical Practitioners Act*, 1938-1939.
Szput, Maurycy, M.D., 1936 (Univ. Paris), 45, Carillon Avenue, Newtown. Registered in accordance with the provisions of Section 17a of the *Medical Practitioners Act*, 1938-1939.

The following additional qualifications have been registered:
Johnson, Alexander Skeffington, Sydney (M.B., B.S., 1933, Univ. Sydney), M.S., Univ. Sydney, 1939.
Marshall, Stuart Vance, Bathurst (M.B., 1925, Ch.M., 1935, Univ. Sydney, D.A. (R.C.P. and S., 1935)), M.R.A.C.P., 1940.
Rose, Thomas Frederick, Brisbane (M.B., B.S., 1933, Univ. Sydney), F.R.C.S. (England), 1939, F.R.C.S., (Edinburgh), 1939, M.R.C.O.G. (London), 1939.
Lawes, Frank Augustus Essery, Hornsby (M.B., Ch.M., 1921, Univ. Sydney), M.R.A.C.P., 1939.
Rae, Robert Keith, Hornsby (M.B., 1915, Univ. Sydney), M.R.A.C.P., 1939.

The following change of name has been registered:
Edelsten-Pope, Jean, Royal Prince Alfred Hospital, Sydney, now Armytage, Jean.

Obituary.

JAMES MANNING RAINBOW.

WE regret to announce the death of Squadron-Leader James Manning Rainbow, of the Medical Branch of the Royal Australian Air Force, who was killed in an accident at Glenbrook, New South Wales, on January 28, 1941.

HENRY JAMES WILLIAMS.

WE regret to announce the death of Dr. Henry James Williams, which occurred on January 26, 1941, at Brunswick, Victoria.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Barrett, Saywell Clifton, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

Corrigendum.

IN the issue of January 25, 1941, there was published on page 121 the abstract of an article by D. Williams and D. Brinton from *The Lancet* of October 19, 1940, on the treatment of cerebro-spinal fever with sulphapyridine. The dose in this abstract was stated as grains instead of grammes.

Medical Appointments.

Dr. M. L. Unwin has been appointed Government Medical Officer at Tully, Queensland.

Dr. J. E. Overstead has been appointed Government Medical Officer at Atherton, Queensland.

Books Received.

"The 1940 Year Book of Pathology and Immunology", edited by H. T. Karsner, M.D., and S. B. Hooker, A.M., M.D.; 1940. Chicago: The Year Book Publishers. Crown 8vo, pp. 688, with illustrations. Price: $3.00 net.

"Syphilis in Earlier Days", by J. R. Whitwell, M.B.; 1940. London: H. K. Lewis. Crown 8vo, pp. 98. Price: 5s. net.

"War Primer on Wound Infection: Its Causes, Prevention and Treatment", by W. H. Ogilvie, Robert Cruickshank, Lawrence P. Garrod, L. E. H. Whitby, G. A. H. Buttle; 1940. London: The Lancet Limited. Demy 8vo, pp. 96. Price: 2s. 9d. net.

Diary for the Month.

FEB. 11.—Tasmanian Branch, B.M.A.: Branch.
FEB. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 14.—Queensland Branch, B.M.A.: Council.
FEB. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 26.—Victorian Branch, B.M.A.: Council.
FEB. 27.—South Australian Branch, B.M.A.: Branch.
FEB. 28.—Tasmanian Branch, B.M.A.: Council.
FEB. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia: all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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